A CONTROLLED STUDY OF THE DENTAL HEALTH OF CHILDREN WITH CONGENITAL CARDIAC DISEASE

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A CONTROLLED STUDY OF THE DENTAL HEALTH OF CHILDREN WITH CONGENITAL CARDIAC DISEASE

BY

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STATEMENT OF AUTHENTICITY

The work presented in this thesis is, to the best of the candidate's knowledge and belief, original, except as acknowledged in the text. The material has not been submitted, either in whole or in part, for a degree at this or any other university.

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PUBLICATIONS ISSUING FROM THE THESIS

A paper from this research project has been submitted for publication in a future issue of the journal, Pediatric Dentistry. It is also intended that this research paper be presented at the 31st annual meeting of the Australian and New Zealand division of the International Association for Dental Research to be held in Brisbane, September 30th - October 2nd, 1991.

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LIST OF ABBREVIATIONS

AMERICAN HEART ASSOCIATION	AHA
AUSTRALIAN DENTAL ASSOCIATION	ADA
ATRIAL SEPTAL DEFECT	ASD
ATRIO-VENTRICULAR	A-V
BACTERIAL ENDOCARDITIS	BE
BRITISH SOCIETY OF ANTIMICROBIAL CHEMOTHERAPY	BSAC
CONGENITAL CARDIAC DISEASE	CCD
DECAYED, MISSING, FILLED PRIMARY TEETH	dmft
DECAYED, MISSING, FILLED PRIMARY TEETH SURFACES	dmfs
DECAYED, MISSING, FILLED PERMANENT TEETH	DMFT
DECAYED, MISSING, FILLED PERMANENT TEETH SURFACES	DMFS
DEVELOPMENTAL DEFECTS OF ENAMEL	DDE
ELECTROCARDIOGRAM	ECG
EUROPEAN SOCIETY OF CARDIOLOGY	ESC
FEDERATION DENTAIRE INTERNATIONALE	FDI
INFECTIVE ENDOCARDITIS	IE
INTELLIGENCE QUOTIENT	IQ
INTERNATIONAL BUSINESS MACHINES	IBM
PERSONAL COMPUTER	PC
STANDARD DEVIATION	SD
STAPHYLOCOCCUS	STAPH
STREPTOCOCCUS	STREP
WORLD HEALTH ORGANISATION	WHO
VENTRICULAR SEPTAL DEFECT	VSD

ABSTRACT

Congenital Cardiac Disease (CCD) represents one of the most common forms of developmental anomalies in children. Affected children require special care in dentistry due to their susceptibility to Infective Endocarditis (IE) from oral infections. Yet little information is available regarding the oral health of children with CCD.

The present study which investigated 39 children with CCD and 33 healthy control siblings, showed that CCD children generally suffered poorer oral health. In patients with primary dentitions, 52 percent of children had developmental enamel defects compared with only 21 percent in the control group ($p < 0.01$). In addition, CCD children had significantly greater numbers of teeth with untreated dental decay (mean dmft 4.2 vs 2.3, $p < 0.01$) as well as greater numbers of endodontically treated teeth. Children with CCD also had less than optimal professional and home care. Only 31 percent have had professional advice regarding the need for increased preventive dental health behaviour and only 15 percent were taking fluoride supplements although residing in a non-fluoridated area. Furthermore, significantly fewer CCD children had parental help with toothbrushing compared with control children ($p < 0.001$). This study shows that children with CCD should be targeted for vigorous dental preventive care.

REVIEW OF THE LITERATURE

INTRODUCTION

Congenital cardiac disease (CCD) represents one of the most common forms of developmental anomalies in children (Behrman & Vaughan, 1983; Nadas, 1984). With increasing sophistication of medical care in recent years, most of these children survive well into adulthood and beyond.

The dental management of children with CCD needs special consideration. Firstly, they are predisposed to develop infective endocarditis (IE) from bacteraemia induced by dental procedures, (Eggleston, 1974; Munroe & Lazarus, 1976; Cawson, 1981) , as well as from chronic poor oral health (Bayliss et al., 1983; Walker, 1984).

Secondly, many of the severely affected children have reduced tolerance to the stress induced by dental treatment (Wright et al., 1985; Rockman, 1989).

Thirdly, complications of CCD such as haematological, respiratory and immunological problems as well as drug interaction with chronic medications must all be taken into account in dental treatment planning of the child with CCD (Hunter, 1974; Elliot, 1975; Hall, 1980).

Yet in spite of the importance of CCD to dental treatment and the significant numbers of children affected, there is a paucity of

information available regarding their dental health and treatment requirements.

To date, only one published survey of children with CCD (Berger, 1978) and one recent case report (Rockman, 1989) have addressed this problem.

In addition, few studies have addressed the problems of dental management in children with CCD. Although recommendations of prophylactic antibiotic cover for dental treatment of CCD patients have been issued by several health authorities - Australian Dental Association (ADA, 1989), American Heart Association (Kaplan et al., 1977; Schulman et al., 1984; Council on Dental Therapeutics, 1991), British Society for Antimicrobial Chemotherapy (BSAC, 1982; BSAC, 1986; BSAC, 1990), European Society of Cardiology (Delaye et al., 1985) and the Medical Letter (1987) - regimes for this are constantly being revised and may vary among different health authorities. Also, dental and medical practitioners may differ in their views of these regimes or may not even use prophylactic antibiotics at all (Oakley, 1987; McGowan, 1987; Kryshtalskyj, 1988; Fekete, 1990).

Furthermore, many types of dental treatment are controversial for patients with CCD, due to the increased risk for IE (Rockman, 1989). These include endodontic treatment for primary teeth and the placement of stainless steel crowns. However, the prevalence of these controversial types of treatment in children with CCD is unknown.

It is thus of great clinical value to examine a group of children with CCD to determine their oral health status, preventive dental health behaviour as well as the nature of previous dental treatment. Results of such a survey would be of value in determining risks of dental disease and treatment of children with CCD as well as providing guidelines to dental practitioners for their dental management.

CONGENITAL CARDIAC DISEASE

CCD comprises one third of all major birth defects. It is characterised by structural abnormalities of the heart or great vessels that actually or potentially interfere with normal cardiac function (Behrman & Vaughan, 1983).

Prevalence of Congenital Cardiac Disease:

Prevalence estimates depend on the definition of the disease and the post natal period when the disease is diagnosed. The first study to ascertain CCD prevalence rates in a community was reported by MacMahon et al. (1953). They estimated that 0.3% of infants born alive have cardiac defects. More recent work by Mitchell et al. (1971) with longer follow up periods have reported that closer to 0.9% of infants are affected with CCD.

Amongst specific cardiac lesions, ventricular septal defects are the most common. Hoffman & Christianson (1978) ascertained the proportion of specific heart defects in their series and compared them with other major studies (Table 1). Among live births the other lesions more commonly diagnosed are patent ductus arteriosus, atrial septal defects, pulmonary stenosis, coarctation of the aorta, tetralogy of Fallot, and transposition of the great vessels.

Demographic Factors:

Maternal age has been reported (Nora & Nora, 1987) as a strong determinant of CCD with a relative prevalence of 1.7 times higher in the offspring of women 40 years or older compared with women less than 40 years of age.

TABLE 1. PROPORTION OF SPECIFIC CARDIAC DEFECTS AMONG LIVE-BORN INFANTS WITH CONGENITAL CARDIAC DISEASE (CCD)

Type of Lesion	Percent*
Ventricular septal defect	30.3
Patent ductus arteriosus	8.6
Atrial septal defect - secondary	6.7
Endocardial cushion defects	3.2
Pulmonary stenosis	7.4
Aortic stenosis	5.2
Coarctation of the aorta	5.7
Transposition of the great arteries	4.7
Tetralogy of Fallot	5.1
Truncus arteriosus	1.0
Hypoplastic left heart	1.3
Hypoplastic right heart	2.2
Single ventricle	0.3
Double outlet right ventricle	0.2
Total anomalous pulmonary venous connection	1.1
Other	17.1

Adapted from Hoffman & Christianson, 1978.
* Based on 3104 cases of cardiac malformations.

While the frequency of CCD overall does not vary according to gender, there is strong evidence that specific lesions are distributed differently between the sexes. Patent ductus arteriosus occurs nearly twice as often in girls, whereas aortic stenosis is three times more common in boys (Rothman & Fyler, 1976). Hypoplastic left ventricle, a defect that may be associated with aortic or mitral stenosis, is also diagnosed more often in male infants.

Maternal Illness and Congenital Cardiac Disease:

Maternal illnesses such as rubella virus infection have been established as causative organisms in the induction of CCD (Tartakow, 1965). Newborns of diabetic mothers have also been observed to have a higher risk of CCD than healthy mothers (Pederson et al., 1964 ; Mitchell et al., 1971).

Principles of Teratogenesis:

The human embryo is most vulnerable to cardiopathic events between the days of germ layer differentiation and completion of cardiogenesis, namely during days 20 through 45 of development. However timing and dose of a teratogenic agent as well as the gestational age of the embryo are too difficult to ascertain with any certainty in epidemiological studies (Harned, 1983).

Drug Teratogens and Congenital Cardiac Disease:

Maternal use of ethanol (Loser & Majewski, 1977), anticonvulsants (Hill et al., 1974), lithium (Schou, Weinstein & Goldfield, 1973), exogenous female hormones (Nora & Nora, 1976) and thalidomide

(McBride, 1961) are among the drugs known to be associated with CCD while salicylate, amphetamines and tranquillisers have been suspected (Turner & Collins, 1975 ; Milkovich & van den Berg, 1977). An antiemetic agent, Doxylamine, has also been suggested to have a teratogenic role in cardiac development from a primate study (Kolata, 1982).

In her recent review of the available epidemiological evidence, Zierler (1985) could not attribute the vast majority of cardiac defects to these cardiac teratogens alone. However, Harned (1983) has cautioned that our understanding of the effects of various deleterious agents on the vulnerable foetus is still very naive. In concluding an extensive review of the aetiology of CCD, he recommends a vigorous effort to understand better the mechanisms of various teratogenic agents and external environmental factors such as pesticides and household toxic substances.

Genetics of Congenital Cardiac Disease:

The genetics of CCD have long been discussed. The known association between CCD and environmental factors such as viruses and the pattern of familial recurrence suggest CCD may be polygenic or multifactorial. At present, it appears that about 8% of cases are predominantly genetic and about 2% are predominantly environmental in aetiology. In the remaining 90% there is an important genetic-environmental interaction (Nora & Nora, 1978).

The results of surveys by Emanuel et al. (1983) and Allan et al. (1986) would support this view, with an increased recurrence rate

with a previously affected child within a family. Burn (1983) concludes that it is still reasonable to give most patients with CCD a 5% recurrence risk for an important cardiovascular malformation in offspring, and to interpret this as a relatively small risk. Where antenatal diagnosis using foetal echo-cardiography is available, it is reasonable to offer this facility in view of the greater importance of genetic factors than previously thought.

A prospective study of 233 women by Whittemore et al. (1982) demonstrated the increased risk of CCD in offspring of mothers who had CCD themselves, a risk not significantly affected by surgery and one that is considered to be of genetic importance.

Extracardiac anomalies occur commonly in children with CCD. In a prospective study to detect malformation patterns, Kramer et al. (1987) noted CCD either occurred alone or was accompanied by a major extracardiac malformation in 7.7% of cases, or occurred as part of a malformation syndrome in 13.3% of cases. It is interesting to note the higher incidence of CCD among children with facial clefting. Up to two-thirds of these children had severe cardiac disease when checked at post mortem (Shah et al., 1970). In living patients with facial clefts, the incidence was much lower at 1.3%. The incidence of cardiac disease, while high (20%) in children dying with the Pierre Robin syndrome was greater (75%) in deceased infants who had cleft palate without the Pierre Robin syndrome.

Similarly, clinic evaluations of facial and cardiac anomalies in DiGeorge syndrome (Van Mierop & Kutsche, 1986), Noonan syndrome

(Noonan, 1968), Velo-Cardiac-Facial syndrome (Shprintzen et al.,1978), foetal alcohol syndrome (Loser & Majewski, 1977) and Williams elfin facies (Jones & Smith, 1975) suggest a linkage for such malformations. This association between cardiac and facial abnormalities in children, when considered with the experimental linkage of neural crest elements to the pathogenesis of conotruncal and facial malformations using chick chimeres as a model (Nishibatake et al., 1987) is thought to occur from failure of the cephalic neural crest cells to translocate and interact properly with the developing embryonic branchial arches. In addition, the severity of the cardiac abnormalities may be related directly to the severity of the associated noncardiac anomalies.

To assess these associations, Bell et al. (1990) studied 20 patients with tetralogy of Fallot or persistent truncus arteriosus using cardiologic, facial dysmorphic and cephalometric criteria. The total group had significantly ($p < 0.00001$) more facial malformations than normal populations while the occurrence rate between the two anomalies was not significant. The tetralogy of Fallot patients exhibited a higher than usual distribution of dolichofacial growth patterns, Class II skeletal relationships, mandibular retrusion and maxillary protrusion. The study also found a higher occurrence of pharyngeal arch derivative malformations in the more severe patent truncus arteriosus group as compared to the tetralogy of Fallot group. This hints that facial dysmorphism is a measure of potential cardiac severity, though selection bias was present in that the study population was limited to survivors of cardiac surgery for conotruncal defects.

Growth and Development of Children with Congenital Cardiac Disease:

Children with CCD may have significant growth disturbance. A longitudinal study by Bayer & Robinson (1969) has shown that children with CCD are smaller than the norm when assessed by five basic body measures, namely weight, height, sitting height, shoulder breadth and pelvic breadth. Girls and boys are relatively smaller than normal children in both decades of life; but girls having approached the norm in the second decade of life. The growth effect of corrective surgery is consistently but only slightly beneficial indicating genetic or environmental factors are more important in growth inhibition.

Linde et al. (1970) studied the effects of CCD on intellectual and emotional development. Cyanotic children showed a lag in intellectual development, particularly in early years, related in part to physical incapacity. IQ increased in operated cyanotic children but not in the inoperable cyanotic group. Children with acyanotic CCD were less incapacitated initially and their original IQ was closer to their true potential; therefore, less change was seen at the time, whether or not a cardiac operation intervened. Greatest improvement in psychological adjustment and behaviour was seen in operated cyanotic children, with less change in the acyanotic children. After operation, mothers of all CCD children were less anxious about them and pampered and protected them less. The data implies a relationship between the magnitude of parental concern and the degree of cyanosis.

Physical ability increased in all children after operation, but more so in those who were cyanotic previously. Physical status did not change in normal children and in unoperated children with heart disease. Overall, physical function scores were slightly below average for the CCD group. This result has been confirmed by a more recent survey (Wright et al., 1985), particularly with regard to muscular endurance and motor ability. Ferenz et al. (1980) found that about 25% of the young adults aged 14 to 21 years with CCD frequently perceived themselves as more limited physically than their medical records would suggest. Childhood activities had been affected in about 30% of cases, but seriously disrupted in only 17%.

There is little indication of problems with school. Most children with CCD had positive experiences and were treated like other children. Wright et al. (1985) found that 68% sought a skilled or profession occupation. Normal social behaviour was much more common than antisocial behaviour. Linde et al. (1970) reported that parental overprotection and childhood behaviour problems went together, although corrective surgery reduced the overprotection and resulted in both better school behaviour and a reduction of home problems. Wright et al. (1985) disagreed in that antisocial scores in their study did not correlate to whether subjects had had corrective surgery. They found that specific problems and generalised health worries were not more usual in those with objective evidence of more serious CCD. Although Offord et al. (1972) indicated that children with CCD felt they were more vulnerable to health problems in general than normal children and that the same would be true in their adult years.

Ferenz et al. (1980) noted in a study of 46 children that knowledge of the heart condition was seldom entirely correct (11%) and most

often was partially correct (76%). However, both groups sought more information on how heart disease may affect their life style.

Diagnosis and Classification of Congenital Cardiac Disease:

In a review of the diagnostic criteria for CCD, Porter (1965) lists such objective symptoms as cyanosis, cardiomegaly, tachycardia, temperature, clubbing of the extremities, right and left ventricular hypertrophy as the most significant.

It is interesting to note that no murmur is present in the newborn infant with a ventricular septal defect. This is because mitral pulmonary hypertension creates equal pressures in right and left ventricles, thereby eliminating turbulent flow until pulmonary pressure falls usually by the fifth day when a murmur becomes audible. Infants with small defects have no symptoms and develop normally while larger defects usually result in the baby becoming breathless and sweaty during feeding at two to three months. Weight gain is slow and the infant is susceptible to chest infection and fails to thrive. The two typical features of a patent ductus arteriosus are collapsing peripheral pulses and a continuous or "machinery" like murmur, while atrial septal defects do not cause symptoms in infancy. Positive clinical findings only occur later in childhood and include a palpable right ventricular impulse, an ejection systolic murmur in the pulmonary area and an associated mid-diastolic tricuspid flow murmur if the shunt is large (Radford, 1989).

The two most common stenotic lesions, pulmonary stenosis and aortic stenosis both feature a harsh ejection systolic murmur in the pulmonary and aortic areas respectively. The lower and longer the murmur, and the later its systolic peak, the more severe is the stenosis (Radford, 1989).

The two remaining cyanotic lesions most commonly encountered, coarctation of the aorta and tetralogy of Fallot both display classical symptoms of cyanosis, cyanotic spells, cardiac failure and finger clubbing. A soft systolic murmur is noted in the former while an ejection systolic murmur is found in the latter (Radford, 1989).

Non-invasive diagnostic methods in paediatric cardiology include postero-anterior chest x-ray silhouette information, electrocardiogram to record electrical activity of the heart during the cardiac cycle and echocardiography to obtain two dimensional ultrasonic sector imaging of the heart and vessels. Utilising the Doppler principle, evaluation of velocity profiles within the heart and great vessels can be made (Goh, 1989).

Invasive diagnostic techniques include first-pass angiography, ECG-gated angiography and myocardial and pulmonary perfusion techniques. Cardiac catheterisation forms the major and most important invasive tool in the hands of the paediatric cardiologist. However, this has been modified by the newer non-invasive techniques. It allows haemodynamic assessment of shunt flow, anatomic evaluation by

cineangiography, biopsy, angioplasty, embolisation procedures and intracardiac electrophysiology studies (Goh, 1989).

As technological advances in paediatric cardiology occur, the accurate rapid diagnosis of CCD for neonates has been possible. Murphy et al. (1985) prospectively studied 104 infants who were younger than seven days of age and in whom CCD was suspected. The presence of CCD was detected non-invasively in 64 infants. The other 40 infants were considered not to have CCD. None of these 40 infants underwent cardiac catheterisation for the purpose of excluding CCD although three (7.5%) were subsequently found to have incidental cardiac lesions. In nine (14%) of the infants with CCD the non-invasive diagnosis was incomplete. The results of this study indicate that CCD can be confidently excluded as a cause of distress in the sick neonate without the use of invasive methods such as catheterisation.

Hence the importance of early recognition of the signs of serious heart disease in order to reduce the level of infant mortality. These signs and symptoms include cyanosis that does not clear with the administration of oxygen, the presence of tachycardia, respiratory distress or hepatomegaly. Rubin et al. (1987) emphasised that these are cardinal signs and symptoms of neonatal heart disease which even in the absence of murmur should lead to swift action, since the condition of infants with cardiac lesions severe enough to cause these findings will rapidly worsen and never improve.

ORAL HEALTH OF PATIENTS WITH CARDIAC DISEASE

Berger (1978) studied the dental health and preventive behaviour of 57 affected children compared to a healthy group and found significant differences between the two groups. Although no DMFT figures were available, the author reported that children with CCD had significantly greater numbers of actively carious primary as well as permanent teeth and a higher prevalence of premature unilateral loss of primary molars. Furthermore, they had a higher prevalence of developmental defects such as enamel hypoplasia and discolourations of the teeth. Also it was of clinical significance to note that within the group of children with CCD, cyanotic children constantly showed greater numbers of carious teeth and developmental defects.

Periodontal Health in Patients with Cardiac Disease:

With regard to periodontal health, Berger's study reported that children with CCD had significantly higher periodontal index scores in relation to their oral hygiene index scores, compared to healthy children, indicating that they may be at higher risk to develop periodontal disease. This may be related to the fact that she reported children with CCD brush less frequently, had fewer professional dental health visits and less parental reminders to brush compared to the healthy group.

The study by Berger has thus shown that children with CCD are at greater risk to develop dental disease which may lead to further serious medical consequences.

Both Valachovic & Hargreaves (1979) and Lampe et al. (1978) have suggested that infected primary molars may be the foci of infection in children with CCD who developed brain abscesses and subsequently died.

The recent case report (Rockman, 1989) of a young child with Tetralogy of Fallot has confirmed the features of dental neglect in children with CCD and the need for comprehensive controlled studies of the oral health status of such children.

Patients undergoing cardiac valve surgery are at high risk of developing IE in the pre-operative and post-operative periods. The level of dental health in this selective group of high risk patients has been found to be very poor (Thom & Howe, 1972; Holbrook et al., 1981; Rogers, 1989). This is in contrast to the widely accepted view that good oral health is essential in the prevention of bacteraemia and subsequent IE.

In the study of 50 adult patients, Thom & Howe (1972) considered only 14 to be dentally fit, 7 of whom were edentulous. Thirty-nine patients sought dental treatment for the relief of pain alone, and their dental condition was far from satisfactory. Less than one quarter of the patients included in this study had been warned of the need for maintenance of a high degree of dental fitness and prophylactic measures before treatment. Forty-three had never had a medical history taken before dental treatment was performed.

Similar findings were reported by Holbrook et al. (1981) with dental infection common in the adult dentate patients; 60% had either chronic periodontal inflammation or an abscess or both. Only 17 of the 42 patients with teeth could be regarded as having satisfactory dental health. Only nine patients with teeth knew of their special need to maintain good dental health.

In contrast to these two surveys, Rogers (1989) studied 31 patients shortly before and after they were due to have cardiac surgery. Although 24 (77%) had no dental symptoms only two were considered dentally fit and 22 (65%) required the extraction of one or more teeth. On average patients had not attended a dental practice for more than nine years. Lack of symptoms was not protection against the presence of a septic focus of dental origin and unfortunately, it is well recognised that many patients only seek a dental opinion when in pain. In this study, the level of disease in asymptomatic patients was high and must raise doubts as to the dental fitness of patients in centres where referral for dental assessment is on the basis of symptoms alone.

Dental Caries in Patients with Cardiac Disease:

Many studies (Bosso & Pearson, 1973; Lokken et al., 1975; Feigal et al., 1981) suggest an increase in cariogenicity due to chronic exposure to sugar containing medications. The only clinical study (Roberts & Roberts, 1981) of caries caused by liquid medication intake previously recorded was done on children from a non-fluoridated community who were taking a variety of medications. In this study, the authors found a greater incidence of dental caries

and gingivitis in children taking medications than in controls. The exact degree of difference is difficult to ascertain since the examinations were done without the aid of radiographs.

In a more detailed study (Feigal et al., 1984) to test the caries effect of one sucrose containing medication, Lanoxin, in young cardiac patients compared with children who had no chronic medication intake, there was a significant difference between the groups. The cardiac group had a mean dmfs of 4.57 compared with 1.55 for the control. There was also a large proportion of patients in the high caries category (> 10 dmfs) for the cardiac group compared to the control.

The multifactorial aetiology of dental caries makes it difficult to derive cause and effect relationships between dental caries and medications from human studies. Many confounding variables may be involved when studying a particular group such as children with CCD. Young cardiac patients may be expected to have lower caries rates because of heightened health awareness and exposure to preventive medicine. On the other hand children with CCD may be a protected and coddled group, factors which may increase consumption of cariogenic snack foods and therefore, dental caries. Definitive studies on these factors in children with CCD do not exist.

Other Aspects of Oral Health in Patients with Cardiac Disease:

Hall (1980) described the first comprehensive studies of dental and oral changes found in children with CCD by Kaner et al. (1946 & 1949) and Hakala (1957). Kaner (1946) compared cyanotic and

acyanotic cardiac and normal children and found in the cyanotic group the following features:

1. Delay in eruption of deciduous and permanent teeth.
2. Higher caries incidence.
3. Histologically, dilated pulp capillaries and irregular odontoblasts and predentine.
4. Cyanosis of oral mucosa, lips and tongue (mild bluish colour to dark brownish red) which returns to normal after cardiac surgery.
5. Marginal gingivitis with bleeding.
6. Teeth look "paper white" against dark blue mucosal/ gingival background.

In addition, Hakala (1957) found:

7. Higher incidence of enamel hypoplasia (incidence being the same in acyanotic and control groups).
8. Histologically, less keratinisation of oral epithelium, enlarged blood vessels of submucosa, abundant inflammatory cells especially perivascularly.
9. High incidence of fissured tongue and geographic tongue.
10. High incidence of position anomalies of teeth.

Abnormal bleeding following dental extractions or surgery is common in children with cyanotic CCD. The coagulation factors in these children were studied by Goldschmidt (1966), who found a reduction of coagulation factor activity related to the increase in haemoglobin and haematocrit values. It is now known that platelet adhesion to collagen is abnormal and probably coagulation factor production also is affected (Nadas, 1984).

DENTAL MANAGEMENT OF CHILDREN WITH CONGENITAL CARDIAC DISEASE

Hall (1967) made the following recommendations in the management of children with CCD undergoing dental treatment:

1. Preventive dental care from birth or diagnosis is essential using all available techniques.
2. Close cooperation between dentist and cardiologist is essential.
3. The dentist must take an adequate medical history which must be updated.
4. Premedication is a good practice for these children as it decreases apprehension and minimises blood pressure rises.
5. Saliva inhibiting drugs should not be used without consulting the physician.
6. Local anaesthesia is the anaesthesia of choice and it is essential to achieve total anaesthesia. The use of topical anaesthesia prior to injection is recommended, and an aspirating syringe should always be used. The smallest amount of solution compatible with total anaesthesia should be injected. The patient should be observed after injection. The use of a synthetic polypeptide vasoconstrictor, Felypressin, may be preferable to the use of adrenaline for routine operative procedures, although Xylocaine may be used for extractions due to its superior haemostatic effect especially in the cyanotic cardiac patient with a coagulation problem.
7. A synthetic vasoconstrictor should always be used for gingival retraction and adrenaline never used.

8. General anaesthesia is frequently indicated for treatment, in which case the anaesthetic should be administered in a paediatric hospital by a specialist paediatric anaesthetist with full oxygenation. The heart action, pulse and blood pressure should be monitored continuously during the procedure.
9. Facilities must exist for management of cardiac arrest and the dental practitioner and his staff should be able to recognise cardiac arrest and know how to act in the event of such an emergency.
10. Haematological examination with coagulation studies should be carried out before extraction or oral surgical procedures in children with severe CCD.
11. Prophylactic antibiotic therapy should be given to all patients with a CCD, with rheumatic carditis or with an A-V shunt or indwelling transvenous cardiac pacemaker and also for renal dialysis patients with implanted arterio-venous shunt appliances.

The early referral for preventive care of babies and infant children with CCD will eliminate the risks which undoubtedly exist for the patient when dental treatment has to be undertaken in an unhealthy mouth. A special coordinated dental service such as described by Elliott (1975) with emphasis on dental prevention and routine follow-up would significantly improve the quality of life for these children, by reducing the need for extensive dental treatment.

INFECTIVE ENDOCARDITIS

Infective endocarditis (IE) or bacterial endocarditis (BE) is an inflammation of the inner lining of the heart. This can be caused by various organisms but more commonly bacteria such as Streptococcus viridans in the subacute form, or Staphylococcus aureus in the acute form, are responsible. A predisposing factor is necessary for this inflammation to occur, usually an underlying congenital abnormality or valvular damage from rheumatic fever previously (Radford, 1989).

Endocarditis leads to the formation of friable vegetations consisting of fibrin, bacteria, red blood cells, white blood cells, platelets and necrotic tissue on the abnormal inner surface. These vegetations may embolise, resulting in skin lesions, glomerulonephritis, renal infarcts, splenic infarcts, myocardial infarction or cerebrovascular accidents. When the right side of the heart is involved, pulmonary emboli may occur with subsequent fatality. The infective process can also lead to ulceration and perforation of cardiac valve leaflets and occasionally to abscess formation or to myocarditis (Radford, 1989).

Prevalence of Infective Endocarditis:

Infective Endocarditis is a serious disease. Despite the amazing progress in modern medicine and the availability of curative antibiotics the mortality rate of IE although less than pre-antibiotics, is still quite high (Dormer, 1958). In cases diagnosed prior to autopsy, the 60 day fatality rate fell from 46% during 1950 through to 1959 to 22% and 26% during 1960 through to 1969 and 1970

through to 1981 respectively (Griffin et al., 1985). The mean annual age and sex adjusted incidence rates per 100,000 person-years were 3.8 for total cases and 3.2 for definite and probable cases only with little reduction in the 30 year period surveyed.

Skehan et al. (1988) conducted a similar survey over a much shorter period and noted similar results. The overall mortality rate was 21%, however this varied considerably among the different groups studied. Infection associated with mitral valve prolapse or CCD had a mortality rate of 7 for those coming to operation and 19% for those treated medically. Prosthetic valve infection had a mortality rate of 53%. Increased age adversely influenced outcome with a 34% mortality rate among patients aged 60 years and over compared with 9.5% mortality rate in those under 60 years.

Aetiology of Infective Endocarditis:

No source of infection was identified in over half the number of cases studied (Skehan et al., 1988). The most frequent single cause for IE was dental treatment within three months of symptoms. This occurred in 14% of patients while 11% had untreated dental disease thought to be the source of infection. Thus dental treatment or disease was implicated in 25% of all cases.

Bayliss et al. (1983) noted that the source of infection is often not known and that the proportion of cases related to dental procedures or sepsis is probably smaller than previously believed. Their study of 541 patients confirmed this in which they stated in 60% of cases the portal of entry of infection could not be

ascertained: 19% were probably of dental origin and 16% arose from the alimentary, genito-urinary or respiratory tracts or from the skin or in association with drug addiction, fractures or pregnancy and the remaining 5% were related to cardiac or other vascular surgery, cardiac catheterisation, haemodialysis or other procedures involving the blood stream.

Interestingly, 43% of patients were not known to have any cardiac abnormality before the onset of IE. Pre-existing rheumatic heart disease was present in 23% and the incidence of congenital heart disease was slightly lower, 18%. IE can arise in previously normal hearts with apparently normal valves apart from the damage caused by the vegetations.

Clinical features of Infective Endocarditis:

The onset of IE is frequently insidious with malaise, lassitude and fever. Often a viral illness or influenza is suspected initially and delays in establishing the correct diagnosis can occur. Signs of the underlying cardiac lesion will be present such as a systolic murmur or a valvular click. Congestive cardiac failure may develop with associated valvular incompetence or myocarditis. Systemic signs of IE include finger clubbing, petechiae especially in the conjunctivae and oral mucosa, splinter haemorrhages under the nails, Janeway lesions (small, non-tender, macular lesions on palms and soles), and Oslers nodes (tender erythematous lesions on palms, soles and tips of fingers and toes). Emboli can cause hemiplegia and other neurological deficits, splenic infarcts with a friction rub, small bowel infarction and haematuria (Radford, 1989).

Diagnosis of Infective Endocarditis:

The most important factor in the diagnosis of IE is a high index of suspicion when unexplained pyrexia develops in a child with CCD. Confirmation of the diagnosis depends on obtaining positive blood cultures. Full blood count is likely to show a normocytic, normochromic anaemia, elevated white cell count and an elevated erythrocyte sedimentation rate. Hyperglobulinaemia and a positive rheumatoid factor are common. Echocardiography may be helpful in identifying the presence of vegetations of moderate size, but their apparent absence does not exclude the diagnosis of endocarditis. Valvular lesions such as paravalvular abscess or complications such as pericardial effusion may be identified by the echocardiogram (Radford, 1989).

Pathogenesis of Infective Endocarditis:

It is almost universally agreed that IE in general and subacute BE in particular are most frequently caused by Strep. viridans (Dormer, 1968; Mostaghim & Millard, 1975; Bayliss et al., 1983; Skehan et al., 1988). It would appear that the acute form is caused by Staph. aureus and that it is decreasing from 42% of cases in one study (Falace & Ferguson, 1975) to 32% recently (Griffin et al., 1981).

As Strep. viridans is the most common commensal organism of the oral cavity and pharynx, circumstantial evidence exists for a dental cause of IE in most cases. Bahn et al. (1978) have been able to induce IE by introducing Strep. mutans and Strep. mitis into the wound socket of an extracted incisor, into the gingiva or by intravenous injection into the marginal ear vein of the rabbit. The

animals were predisposed to IE by sterile heart catheterisation. The induced endocardial lesions were reported histologically similar to those observed in human IE. McGowan & Hardie (1974) also produced IE in rabbits following dental manipulations after inducing sterile endocardial lesions. However, Cameron (1971) has recorded a case of subacute BE occurring in an edentulous patient whose edentulous state was confirmed by full mouth radiography.

The other concept that links dental treatment to IE is that transient bacteraemias occur frequently after virtually every dental procedure including toothbrushing (De Leo et al., 1974; Faigel & Gaskill, 1975). Bacteraemias are more likely to be heavy and to follow operative damage to the periodontal tissues as a result of extractions (Burket & Burn, 1937; Jones et al., 1970), deep scaling and periodontal surgery (Lineberger & De Marco, 1973). These bacteraemias are said to be of varying duration ranging from five minutes to several hours after the particular procedures. Compared to tooth extraction and gingival manipulation the concentration of organisms entering the dental pulp would appear to be much less following pulpotomies. The one transient bacteraemia found in the 25 cases studied by Farrington (1973) would indicate that approximately 4% of the instances of formocresol pulpotomy would result in a transient bacteraemia. This could be due to the fact that the remaining pulp tissue is covered with formocresol, a highly bactericidal agent, and is never exposed to the oral fluids in which these organisms are found. After extraction, periodontal therapy or luxation of teeth the ruptured vessels form an open pathway from the oral cavity into the blood stream.

The evidence that dental manipulation is a common cause of IE is therefore tenuous and both Guntheroth (1984) and Pogrel & Welsby (1975) go so far as to state that it would appear that dental factors, particularly actual dental treatment, may be less important in the aetiology of IE today than many previous studies have suggested.

However, the possibility remains that dental sepsis can precipitate IE in other ways. Many papers as reviewed by Cawson (1981) have shown that bacteraemias can follow the most trivial of procedures; including brushing the teeth and using an oral irrigating device. Paradoxically, the possibility that dental sepsis can precipitate IE without operative intervention makes the prevention of this disease even more difficult. Thus it has, reasonably enough, been suggested by Dormer (1958), Waddy (1973), Pogrel & Welsby (1975), Bayliss et al. (1983) and Guntheroth (1984) that an important factor in the prevention of IE would be the maintenance of high standards of oral and dental hygiene. Some, however, might question such a contention in view of the fact that, as mentioned earlier, oral hygiene procedures can cause bacteraemia and occasionally IE. If such a suggestion seems perverse, it is none the less important to appreciate that the very frequency with which bacteria are released from around the teeth makes the total prevention of dentally associated IE an impossibility in that it is clearly impractical to suggest that antibiotic cover be given on every occasion when a bacteraemia may be expected, such as toothbrushing or even before meals. This consideration has, however, important implications for practical prophylactic procedures to be discussed later.

Treatment of Infective Endocarditis:

To be effective treatment should be commenced promptly, the responsible organism identified and the appropriate bactericidal antibiotic administered. This is usually given parenterally in high doses and continued for long enough to effect a cure, usually four to six weeks. Bed rest to decrease workload on the myocardium is essential.

Cardiac surgery may become necessary and can be a life saving procedure if there is an acute valvular regurgitation with cardiac failure, if the infection is unresponsive to chemotherapy or if there is embolisation from valvular vegetations. Patients should be examined and reviewed frequently throughout treatment for evidence of changes necessitating surgery (Radford, 1989).

Animal Models of Infective Endocarditis:

Researchers have used animal models to explore the pathogenesis of endocarditis and its prevention by antibiotic administration. Because laboratory animals are highly resistant to the spontaneous development of IE, a mechanism for the reproducible establishment of endocarditis in rabbits and rats has been developed (Glauser & Francioli, 1987). A plastic catheter introduced percutaneously is advanced via the vena cava or the aorta to rub against the tricuspid or aortic valve, respectively. This produces uninfected deposits of fibrin and platelets on the valve leaflets. Subsequently, direct intravascular injection of bacteria or manipulation of oral surfaces produces bacteraemia, and the animals develop valvular lesions which resemble morphologically and histologically those of human IE.

Supporters of this research recognise the limitations of the model. However, most recommendations for prophylaxis stem from some aspects of this system. It was initially observed that bactericidal antibiotic combinations such as penicillin and streptomycin were superior in the prevention of IE in rabbits to agents that were bacteriostatic such as tetracycline (Durack & Petersdorf, 1973; Durack et al., 1974; Southwick & Durack, 1974; Pelletier et al., 1975). It was necessary to use high doses of antibiotics and to maintain high serum and tissue levels for several hours to prevent valve infection. When this data was extrapolated to humans, recommendations entailed multiple doses of antibiotics administered parenterally (Kaplan et al., 1977).

This model of infection is very stringent. More recent observations in animals suggest that if antibiotics are present at the time of induction of the bacteraemia, sublethal and even subinhibitory concentrations of antibiotics were sufficient to prevent IE from developing (Francioli & Glauser, 1985). A reduction in bacterial inoculum by several logs sufficient to produce endocarditis in more than 90% but less than 100% of test animals was believed to make the model more comparable to human infection.

An adaptation of the rat model has been developed to mimic the mechanism of endocarditis that follows dental work. Gingival irritation and a high sucrose diet were used to produce periodontal disease (Overholser et al., 1987; Moreillon et al., 1988). In the presence of a catheter crossing the aortic valve dental extractions were performed. All rats had bacteraemia and 90% developed

endocarditis. The organism that caused endocarditis was not usually the predominant one found during the procedure or the organism most often cultured from the peripheral blood samples taken during the extractions. The parameter which best predicted the likelihood of producing IE among isolates of bacteria from the blood after dental extractions was the in vitro stickiness of a given bacterial strain, not the number of bacteria circulating one minute after extraction. This is similar to the situation in human disease in which the predilection for valve adhesion seems more important in the pathogenesis of endocarditis than mere presence in the blood (Gould et al., 1975).

Thus animal systems may shed some light on structural and evolutionary aspects of IE that cannot be studied in humans. But the determination on how to prevent valve infection by the use of prophylactic antibiotics is dependant on the interpretation of many subtle aspects of the model, and appropriate extrapolation to humans may be difficult.

ANTIBIOTIC PROPHYLAXIS OF INFECTIVE ENDOCARDITIS

The morbidity and mortality associated with IE make preventative therapy attractive. Because dental procedures have long been associated causally with this infection, many prophylactic recommendations have been advised around the time of these procedures. Yet after 30 years of clinical experience and many experimental studies on animals, the benefits of antibiotic prophylaxis have not been conclusively demonstrated. Despite the availability and widespread use of antibiotics, the incidence of IE has not been reduced (Skehan et al., 1988).

Expert committees at the American Heart Association (AHA), Medical Letter, British Society of Antimicrobial Chemotherapy (BSAC) and the European Society of Cardiology (ESC) have devised comprehensive endocarditis prophylaxis strategies. These include dosage recommendations for adults and children, schedules for patients who are allergic to penicillin and recommendations for other procedures involving the genito-urinary and lower alimentary tract as well as for dental procedures. For the sake of simplicity, only recommendations for dental procedures will be reviewed.

The AHA has devised three strategies since 1977 (Kaplan et al., 1977; Schulman et al., 1984; Council on Dental Therapeutics, 1991). The former represents the more conservative view. For adults with CCD, rheumatic or other acquired valvular heart disease, 1.6 million units of intramuscular penicillin or two grams of oral phenoxymethyl penicillin (penicillin V) is given 30 to 60 minutes prior to the procedure, and 500 milligrams of penicillin V is given orally

every six hours after the procedure for a total of eight doses. For children less than 60 pounds, the dosage is a third of the intramuscular dose for adults and 250 milligrams of penicillin V orally every six hours for eight doses.

The second strategy was published in 1984. It calls for two grams of oral penicillin V one hour before the time of the procedure and one more gram six hours later for patients with CCD, rheumatic and other acquired valvular dysfunction undergoing dental procedures that cause gingival bleeding. Pediatric doses were halved for children less than 60 pounds but timing was the same as for adults.

The Medical Letter (1987) has recommended a schedule very similar to the 1984 AHA strategy. The only difference is the lack of a second dose for the parenteral schedule. There is also more flexibility about when to use oral versus parenteral therapy.

A working party of BSAC determined that three grams of amoxycillin taken orally one hour before the dental procedure was suitable for all patients who need prophylaxis (Working Party BSAC, 1982). For children under 10, half the adult dose; for children under five, a quarter of the adult dose. Amoxycillin is better absorbed, reaches a higher serum level and has a longer half-life than penicillin V (Shanson et al., 1978; Shanson et al., 1980). Thus there was no need for a second dose or a parenteral schedule.

Patients who are to have a general anaesthetic should not take drugs by mouth in the preoperative period. Thus any prophylactic

antibiotic must be given by injection. Since one gram of amoxycillin is the maximum intramuscular dose, a second dose should be given to sustain a high blood level. Therefore, at risk patients who are to have a general anaesthetic should receive one gram amoxycillin intramuscularly in 2.5 mls. of 1% lignocaine hydrochloride before induction and a further 0.5 grams amoxycillin by mouth six hours later. Children under 10, half the adult doses of amoxycillin are recommended.

Patients who have already been given penicillin may harbour in their mouths organisms which are more resistant to penicillin than would otherwise be the case (Garrod & Waterworth, 1962). Therefore, it is reasonable to recommend an antibiotic other than penicillin for prophylaxis in such circumstances. Adults who have had penicillin in the preceding month and who would otherwise be given penicillin as prophylaxis should be given erythromycin stearate 1.5 grams orally under supervision one to two hours before treatment, followed by a second dose of 0.5 grams six hours later. For children under 10 half the adult dose; for children under five a quarter the adult dose is recommended.

All the schedules called for erythromycin as the first line oral therapy for penicillin allergic patients. AHA (1984) recommends oral erythromycin one gram (20 milligram/kilogram for children) one hour prior to the procedure, and then 500 milligrams (10 milligram/kilogram for children) six hours later. For patients unable to tolerate oral erythromycin, an oral cephalosporin (1 gram one hour prior to the procedure plus 500 milligrams six hours later)

may be useful, but data is lacking to allow a specific recommendation of this regimen. Tetracyclines cannot be recommended for this purpose. Vancomycin 1 gram for adults (20 milligram/kilogram for children) administered intravenously slowly starting one hour prior to the dental procedure is recommended for high risk patients allergic to penicillin (AHA, 1984). Because of the long half-life of vancomycin, a repeat dose should not be necessary.

The working party of BSAC (1982) recommended in general dental practice adults allergic to penicillin be given 1.5 grams erythromycin stearate orally under supervision one to two hours before the dental procedure, followed by a second dose of 0.5 grams six hours later. For children under 10, half the adult dose; for children under 5, a quarter the adult dose is recommended. Since 1982, reports (Poswillo, 1988) of nausea after the 1.5 gram dose have been made. The amended recommendations (BSAC, 1990) now include a clindamycin schedule as an alternative to the erythromycin regimen. For adult patients, 600 milligrams as a single oral dose taken under supervision one hour before the dental procedure is recommended. For children under 10, 6 milligram/kilogram single oral dose taken under supervision one hour before the dental procedure is recommended. In the BSAC report (1986), adults who are allergic to penicillin and who must have a general anaesthetic were recommended vancomycin 1 gram by slow intravenous infusion over 60 minutes followed by gentamicin 120 milligrams intravenously immediately before induction of anaesthesia. This recommendation has remained unchanged in the 1990 update.

Emphasis of the need for regular dental attendance for the maintenance of good oral health for patients susceptible to IE is made in all the various schedules.

Current strategies favour the use of oral route of bactericidal doses of antibiotics during the procedure to prevent bacteraemia. However, perfect compliance with these recommendations is still unlikely for several reasons. Firstly, universal voluntary compliance is seldom achieved among health care professionals (Brooks, 1980; Murrah et al., 1987; Gutschik & Lippert, 1989). Secondly, there is a problem identifying all the patients who may be at risk, as many do not know they have a heart problem and others mistakenly believe that they do (Lockhardt et al., 1989). Thirdly, patients may forget or refuse their medication. Even with patient selection, there will be instances where antibiotics fail (Durack et al., 1983; Denning et al., 1984) or an adverse effect of the drug outweighs the potential benefit.

Yet because of patient expectations and a widely accepted standard of care promoted by most professional dental associations, much effort will be expended to prevent those few cases of IE that currently result from dental procedures.

The controversy over which antibiotic schedule to use seems to be resolving itself, as the major recommended protocols are comparable (BSAC, 1990; AHA, 1991). In recent reviews of endocarditis prophylaxis, Oakley & Sommerville (1981), Hills-Smith & Schuman (1983), Dajani (1985), Kaye (1986), Farber (1987) and Rakes et al.

(1988), emphasise the similarities of the most recent protocols and discuss the importance of education and implementation.

Recent surveys (Levers & Scully, 1986; Holbrook et al., 1987; Gould, 1990) have highlighted the wider acceptance of the simpler and more practical recommendations now in use. This contrasts sharply with the confusion and the lower compliance rates of previous studies (Durack, 1975; Hashway & Stone, 1982; Holbrook et al., 1983; Gould, 1984; Sadowsky & Kunzel, 1984). Prophylaxis for IE would appear to be widely practised and the recommendations are generally accepted but there is still room for improvement in practice, particularly in drug dosing, timing and duration of prophylaxis.

Alternatives to prophylactic antibiotics:

There are no published studies that report the experience of dental procedures performed on patients with CCD in whom prophylaxis was deliberately withheld. One study (Tzukert et al., 1986) reported the use of a protocol for oral hygiene that included antimicrobial oral rinses such as 0.5% chlorhexidine before and after dental work, oral health maintenance (fluoridation, frequent visits and so forth), separation of procedures by at least two weeks and close adherence to AHA antibiotic guidelines. There were no cases of IE in 3,400 dental procedures that the authors claimed was less than the expected three to six cases in patients receiving antibiotics alone ($p = 0.0013$). This estimate is speculative, and it is difficult to determine which, if any, of these measures were responsible for the good outcome.

Another way of looking at this issue is to consider those patients who developed IE without having dental procedures. Guntheroth (1984) analysed 18 cases of IE in pediatric patients, nine of which were caused by oral streptococci. These young patients had good oral hygiene and had no occasion to go to the dentist. Only four had caries or periodontal disease at the time of admission for IE. Given the knowledge that bacteraemia accompanies mastication, tooth brushing, and occurs randomly during episodes of oral sepsis, Guntheroth (1984) calculated that bacteraemia is present 179 minutes a day in patients with oral sepsis. In a trip to the dentist, there is a 40% chance of bacteraemia, which lasts for 15 minutes. Thus, in a hypothetical month which ends with a dental procedure, cumulative exposure to the bacteraemia of extraction is six minutes, and 5,370 minutes of "physiologic" bacteraemia. However, Guntheroth (1984) does not advocate abolishing prophylaxis but rather focusing more attention on dental hygiene and prevention of lesions that permit recurrent, random bacteraemias for which prophylaxis is impossible.

Risk-benefit analysis of prophylaxis:

There are two published decision analyses which consider the risks and benefits of antibiotic prophylaxis for people with mitral valve prolapse. Clemens & Ransohoff (1984) determined that parenteral penicillin as recommended by AHA (1977) given around the time of dental procedures would have a higher likelihood of fatal outcome than no prophylaxis. For oral penicillin, the risk of a fatal outcome is lower. However, oral penicillin appears to spare life only in older adults and at an extremely high cost.

Bor & Himmelstein (1984) calculated that in 10,000,000 procedures performed without prophylaxis in patients with mitral valve prolapse, 47 cases of IE would be likely to occur with two fatalities. However, the use of penicillin prophylaxis could lead to 175 fatalities from drug reactions and five cases of IE from antibiotic failure. Thus, they concluded that antibiotic prophylaxis is likely to have a net harmful effect on this patient population.

Even though no data is available on the higher risk conditions such as CCD, these papers both illustrate that the patients' risk of fatal IE is comparable with the risk of dying from allergic reactions to antibiotics, particularly penicillin. Many experts believe that oral dosing is less likely than parenteral to have life threatening allergic sequelae. Minor reactions such as rash, serum sickness and immune haemolytic anaemia are very rare after a single dose of penicillin and they are self limiting.

The administration of antibiotics alters the bacterial ecology of the alimentary tract. In one study (MacFarlane et al., 1983) of bacterial isolates from blood and from dental plaque in healthy patients not receiving antibiotics, all streptococci were highly penicillin-susceptible, and very few were erythromycin-resistant. However, in a study (Leviner et al., 1987) of healthy volunteers given a small amount of penicillin, streptococci resistant to penicillin emerged within hours and persisted up to nine days. This has lead to the suggestion to separate visits by at least 10 day intervals if multiple procedures are intended.

PATIENTS AND METHODS

PATIENTS

The patients were recruited from the Pediatric Cardiology Clinic at The Prince Charles Hospital, Chermside. This study was done in collaboration with Dr D. Radford, Deputy Director of Cardiology, who referred suitable patients to Dr K. Hallett, at the Dental School for assessment. Dr Radford had no knowledge of the oral health of the referred patients.

All children, aged between 3-16 years, who attended the Pediatric Cardiology Clinic and resided in the greater Brisbane area were invited to participate in the study. After Dr Radford explained the importance of a thorough dental assessment in relation to their cardiac condition, an introductory letter (Appendix 1) and an appointment time were forwarded to their address. Forty two patients were approached in this manner and thirty nine consented to participate in the study, giving a participation rate of 93%.

CONTROL PATIENTS

A sibling if present, similar in sex where possible and closest in age to that of the patient was also invited to attend for dental assessment. Thirty three were found to be suitable and included in the study.

DENTAL HISTORY

Detailed dental histories were obtained from a parent with regard to past dental treatment provided, form of management, complications of

treatment and regimes of antibiotic cover prescribed. In addition, details of preventive dental health behaviour such as oral hygiene practices, fluoride history and diet evaluation were obtained. These were recorded in the dental history section (II) of the examination form (Appendix 2).

MEDICAL HISTORY

Relevant maternal, perinatal, neonatal and postnatal histories were obtained from a parent and recorded in the medical history section (I) of the examination form (Appendix 2). In addition , further details of the cardiac condition of each patient were obtained from the Pediatric Cardiology Clinic after the dental examination.

DENTAL EXAMINATION

Dental examinations were performed in a routine manner at the University of Queensland Dental School, and the results recorded on the comprehensive examination data section (III) of the examination form (Appendix 2). Extra oral and intra oral abnormalities of the hard and soft tissues were noted. The teeth were dried and examined with a mirror and dental probe for the presence of dental caries, developmental defects in tooth number, size and form and previous dental restorations. Dental caries was recorded using WHO criteria (World Health Organisation, 1987). Enamel defects were classified according to the DDE (Developmental Defects of Enamel) Index as proposed by the Commission of Oral Health, Research and Epidemiology of the F.D.I., 1982. Agenesis and irregularities of crown form of the teeth were also recorded on the examination form (Appendix 2).

The oral hygiene indices were obtained using a standard periodontal probe to detect the presence of supragingival plaque without trauma to the gingival tissues. The modified plaque index of Loe and Silness (Wei & Lang, 1981) was used, where a plaque score (0 for absence, 1 for presence) was recorded on three surfaces of six individual teeth (16/55, 21/61, 24/64, 36/75, 41/81, 44/84). The plaque index was derived as total plaque score divided by total number of surfaces examined. As gingival probing without prophylactic cover may lead to bacteraemia, no gingival indices were recorded.

The occlusion of the patient was recorded using the Angle classification of molar relationship, and the degree of anterior overbite was recorded as a percentage of complete overbite.

The presence of crowding as noted clinically by lack of space for any erupting permanent teeth was recorded on the examination form (Appendix 2).

Bitewing radiographs and orthopantomographs were taken for those patients above the age of five years to aid in the diagnosis of various abnormalities. Parental consent was sought prior to the taking of all radiographs.

The bitewing radiographs were used in the diagnosis of interproximal caries and to assess the nature of previous restorative dental treatment, particularly with regard to endodontic therapy. Where pulp pathology was suspected periapical radiographs were taken to confirm the diagnosis. The orthopantomographs were used to aid in the

diagnosis of hypodontia, supernumerary teeth, microdontia, macrodontia and taurodontia. Abnormalities of crown root ratio using the measurement criteria and formula as proposed by Lai & Seow, 1990 were carried out from the orthopantomographs.

At the completion of the examination, the parent was advised of the clinical findings and the treatment needs, if any, were discussed. Restorative and preventive dental care were offered to those children who did not have their own dentist or relevant records were forwarded to their respective dental care providers.

DIET HISTORIES

A three day diet history chart (Appendix 3) was supplied to each patient to fill out for three typical days of the week. The days had to be consecutive and include one weekend day. When completed, the diet history charts were returned to the Dental School in stamped self-addressed envelopes to the author. The dietary chart was then analysed with regard to the number of daily sugar frequencies in both solution and retentive forms, and whether the sugar exposures occurred between or during meal times.

ANALYSIS OF RESULTS

1. The oral health of children with CCD is compared with the control children with regard to:

    1.1 Prevalence of developmental anomalies such as enamel hypoplasia, enamel opacity, hypodontia, and the presence of supernumerary teeth.

    1.2 Type of occlusion and presence of crowding.

1.3 Prevalence of treated and untreated dental caries.

1.4 Oral hygiene indices.

1.5 Types of previous dental treatment, particularly endodontic therapy.

2. Preventive dental health behaviour of children with CCD is compared with control children by analysing:

2.1 Frequency of tooth brushing and flossing.

2.2 Fluoride history.

2.3 Frequency of daily sugar exposure.

3. The use of prophylactic antibiotic cover for previous dental treatment in CCD children is analysed with regard to:

3.1 Type of antibiotic used.

3.2 Dosage prescribed.

3.3 Route of administration.

3.4 Timing of administration.

STATISTICAL ANALYSIS

A parametric test, the independent Student's t-test was used for statistical analysis of the data. This was used to test the significance of comparing means and proportions of patients affected in each group; Acyanotic, Cyanotic, Total Cardiac and Control. All mathematical calculations were carried out using Kwikstat, a commercial statistical software package for IBM compatible PC's. A p value of less than 0.01 was considered significant in all statistical calculations and was recorded in the appropriate table, otherwise a non significant (NS) level was given.

RESULTS

DEMOGRAPHIC DATA

The demographic details of the CCD and control patients are shown in Table 2. Altogether 39 patients (19 males, 20 females; mean age 7.5 ± 4 yrs, range 2.8 - 15.0 yrs) with congenital cardiac defects and 33 control siblings (22 males, 11 females; mean age 8.6 ± 3.9 yrs, range 1.0 - 15.0 yrs) were available for study. As shown in the Table, there were no significant differences between the CCD and control patients with regard to age, sex distribution, birth weight and gestational ages.

CARDIAC DEFECTS

Table 3 shows the prevalence of the various classes of cardiac defects in the CCD patients. The defects can be subdivided into acyanotic and cyanotic groups depending on clinical presentation. The underlying cardiac defect may be a singular or multiple lesion and therefore a patient may be included in both categories. This is shown in the Table by 22 patients classified as acyanotic and 17 patients as cyanotic. However, some of these patients have multiple lesions and the total number of lesions in the acyanotic group is 25 and in the cyanotic group is also 25. The most common lesion in the acyanotic group was ventricular septal defect (44%) while transposition of the major vessels (40%) was the most common cyanotic defect.

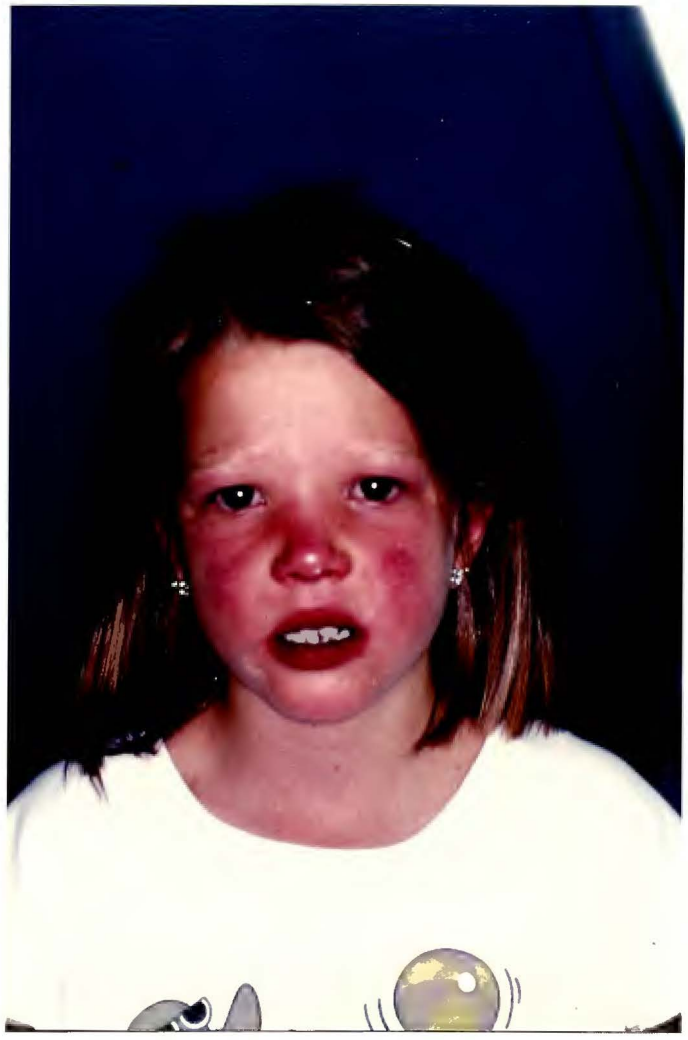

Illustration 1. Typical facial appearance of a CCD patient showing cyanotic features.

TABLE 2. DEMOGRAPHIC DATA OF CCD AND CONTROL PATIENTS

Group	CCD n = 39	Control n = 33	p value
Age: (yrs.)			
(mean ± SD)	7.45 ± 4.00	8.62 ± 3.89	NS
Range	2.83 - 15.00	1.00 - 15.00	
Sex: no. (%)			
Males	19 (49)	22 (67)	NS
Females	20 (51)	11 (33)	
Birth weight: (gms.)			
(mean ± SD)	3155 ± 686	3165 ± 579	NS
Gestational age: (wks.)			
(mean ± SD)	39.4 ± 2.2	39.5 ± 2.2	NS

NS = NOT SIGNIFICANT
There were no significant differences between CCD and control patients with regard to age, sex, birthweight and gestational age.

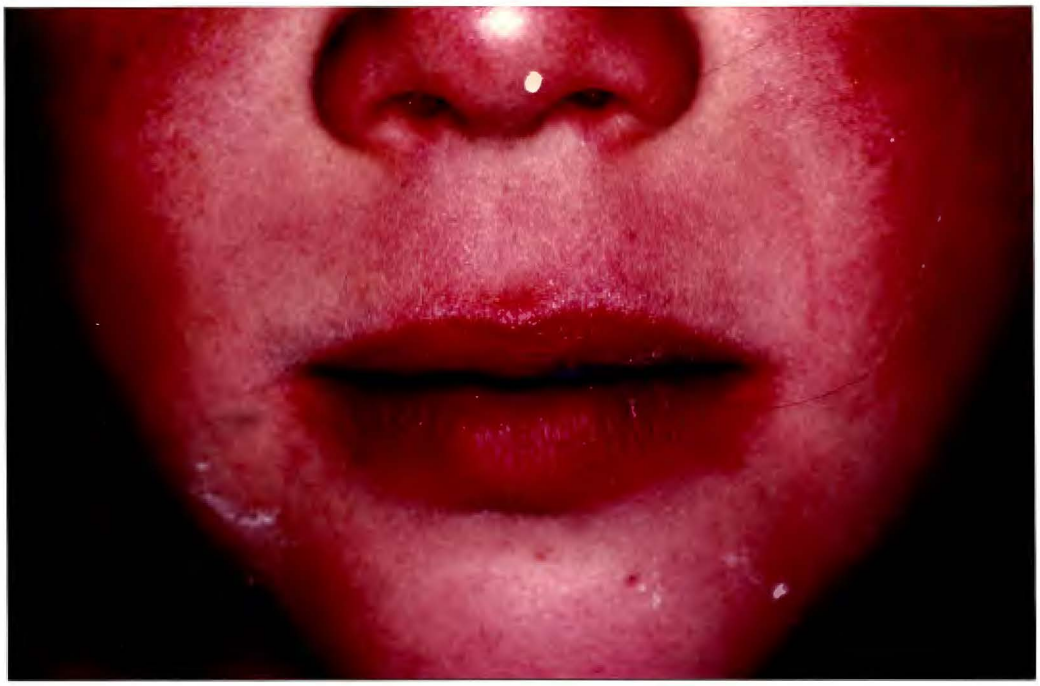

Illustration 2. Cyanotic appearance of lips of a CCD patient.

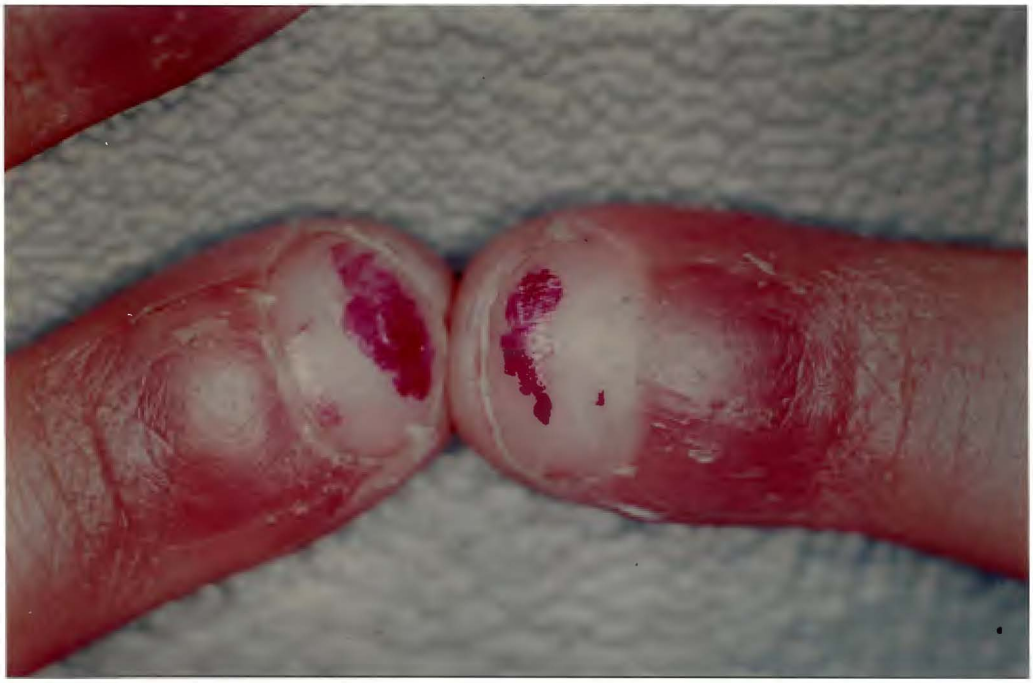

Illustration 3. Finger clubbing indicating development of congestive heart failure in a CCD patient.

TABLE 3. PREVALENCE OF VARIOUS CLASSES OF CONGENITAL DEFECTS IN CCD PATIENTS IN THE STUDY

Cardiac defect n = 39	Number	(%)
ACYANOTIC n = 22		
1. ASD	3	(12)
2. VSD	11	(44)
3. TRUNCUS ARTERIOSUS	2	(8)
4. CARDIOMYOPATHY	2	(8)
5. PULMONARY ATRESIA	5	(20)
6. AORTIC STENOSIS	2	(8)
TOTAL	25 *	(100)
CYANOTIC n = 17		
1. TETRALOGY OF FALLOT	3	(12)
2. EBSTEIN ANOMALY	2	(8)
3. TRANSPOSITION OF MAJOR VESSELS	10	(40)
4. MITRAL ATRESIA	2	(8)
5. TRICUSPID ARTRESIA	2	(8)
6. PULMONARY STENOSIS	6	(24)
TOTAL	25 *	(100)

* some patients have more than one cardiac defect and have been included in several categories

PERINATAL MORBIDITY

The prevalence of various antenatal and perinatal complications and birth history were studied and the details are shown in Table 4. Antenatal complications included maternal hypertension, toxaemia and infection during pregnancy. There were found to be significant differences ($p < 0.01$) between the CCD (44%) and control (12%) patients with regard to these factors. Perinatal complications included a history of respiratory distress, intubation to assist breathing, surgical procedures, hyperbilirubinaemia, hypocalcaemia, infection and failure to thrive in the neonatal period. Only with regard to neonatal surgery was there found to be a significant difference ($p < 0.01$) between the CCD group (33%) and the control group (6%). The birth history was recorded as being a normal vaginal delivery, forceps delivery or caesarean section. No significant differences were noted between the two groups with regard to birth history.

PLAQUE INDICES

As shown in Table 5, the plaque indices at examination for the CCD and control patients were recorded. Mean plaque index for the CCD patients was 0.65 ± 0.2 and 0.58 ± 0.2 for the control patients. Although the mean plaque index was lower for the control group, this was found not to be significant. However, the percentage of patients in the plaque index ranges 0.33 - 0.66 and 0.67 - 1.00, were significantly different between the CCD and control patients ($p < 0.01$). This is illustrated graphically in Figure 1. Here the percentage of CCD patients in the high plaque index group (55%) is significantly higher ($p < 0.01$) than the percentage of control patients in the same plaque index group (24%).

TABLE 4. PREVALENCE OF PERINATAL MORBIDITY IN CCD AND CONTROL PATIENTS

Group	Number of patients / (percentage of group) Acyanotic n=22	Cyanotic n=17	Total CCD n=39	Total Control n=33	p value
Antenatal complications*:	10 (45)	7 (41)	17 (44)	4 (12)	< 0.01
Perinatal complications:					
Respiratory Distress	2 (9)	1 (6)	3 (8)	2 (6)	NS
Intubation	5 (23)	7 (41)	12 (31)	5 (15)	NS
Surgery	3 (14)	10 (59)	13 (33)	2 (6)	< 0.01
Hyperbili-rubinaemia	7 (32)	1 (6)	8 (21)	8 (24)	NS
Other#	0	8 (47)	8 (21)	5 (15)	NS
Delivery:					
Normal	14 (64)	6 (35)	20 (51)	17 (52)	NS
Caesarean	7 (32)	7 (41)	14 (36)	8 (24)	NS
Forceps	1 (5)	4 (24)	5 (13)	8 (24)	NS

* These include Hypertension, Toxaemia, Infection. # These include Failure to Thrive, Infection, Hypocalcaemia.
NS = NOT SIGNIFICANT.
The only significant differences between CCD and control patients were in regard to antenatal complications and neonatal surgery.

TABLE 5. PLAQUE INDICES AT EXAMINATION FOR CCD AND CONTROL PATIENTS

	Number of Patients				
Group	Acyanotic n=22	Cyanotic n=17	Total CCD n=39	Total Control n=33	p value
Plaque index:					
< 0.33	1	1	2	4	NS
0.33-0.66	10	6	16	21	< 0.01
0.67-1.00	11	10	21	8	< 0.01
Mean ± SD	0.67±0.2	0.58±0.2	0.65±0.2	0.58±0.2	NS

NS = NOT SIGNIFICANT
There were significant differences between the proportions of CCD and control patients with regard to plaque indices in the index range 0.33 - 1.00. The range 0.33 - 0.66 had significantly more proportions of control patients while the range 0.67 - 1.00 had significantly more CCD patients.

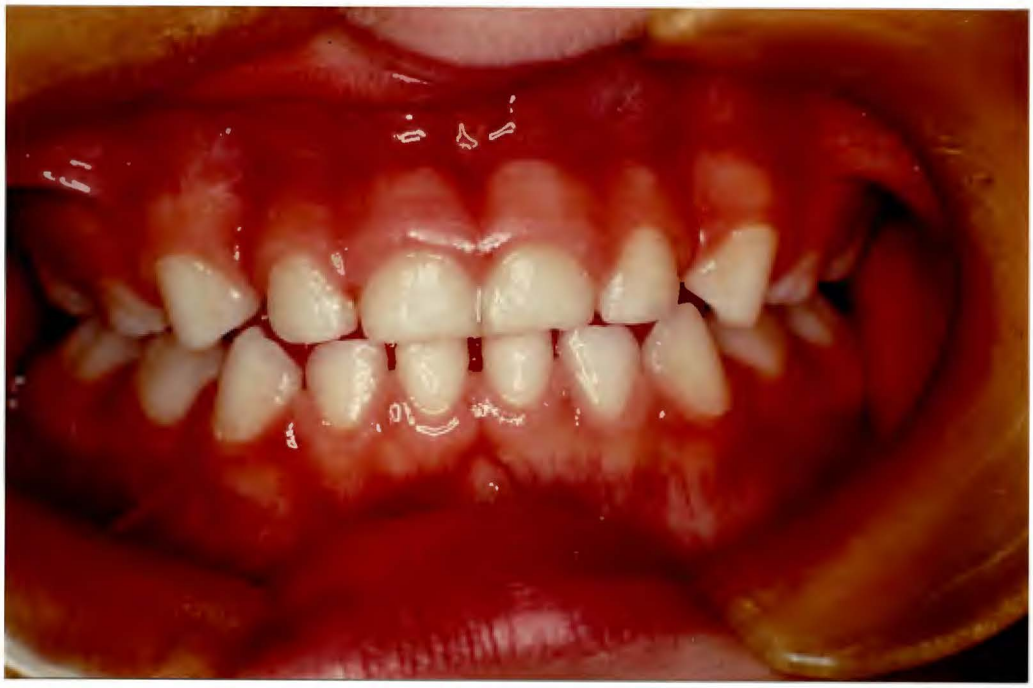

Illustration 4. Appearance of typical primary dentition and gingivae of a Control patient showing low plaque levels, no tooth staining and healthy gingival tissues.

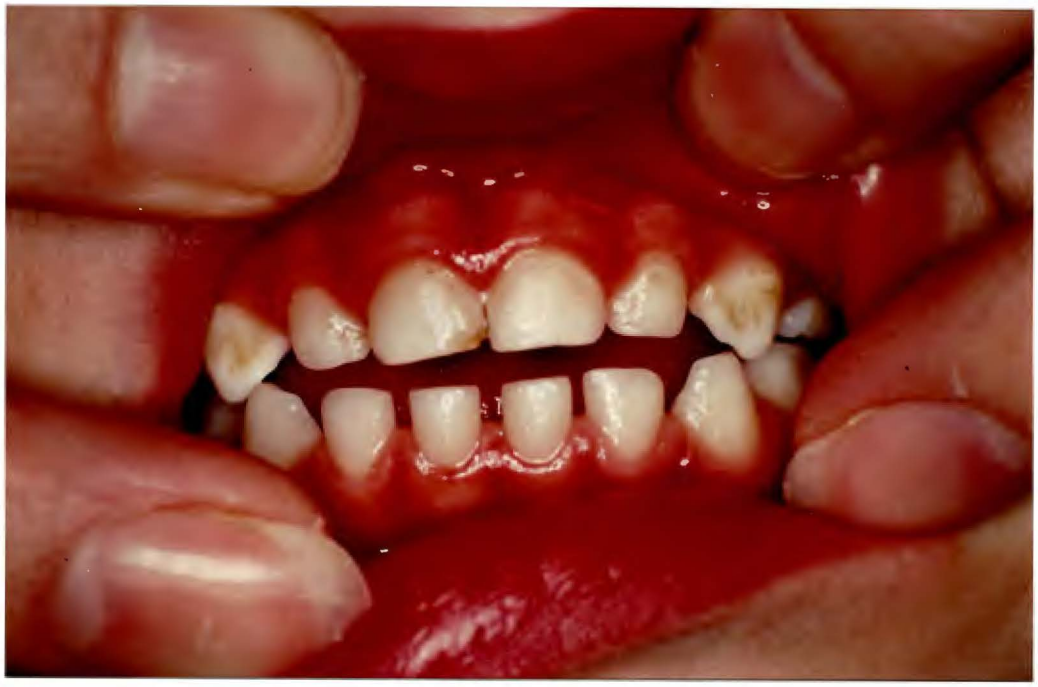

Illustration 5. Appearance of typical primary dentition and gingivae of a CCD patient showing high plaque levels, tooth decalcification and marginal gingivitis.

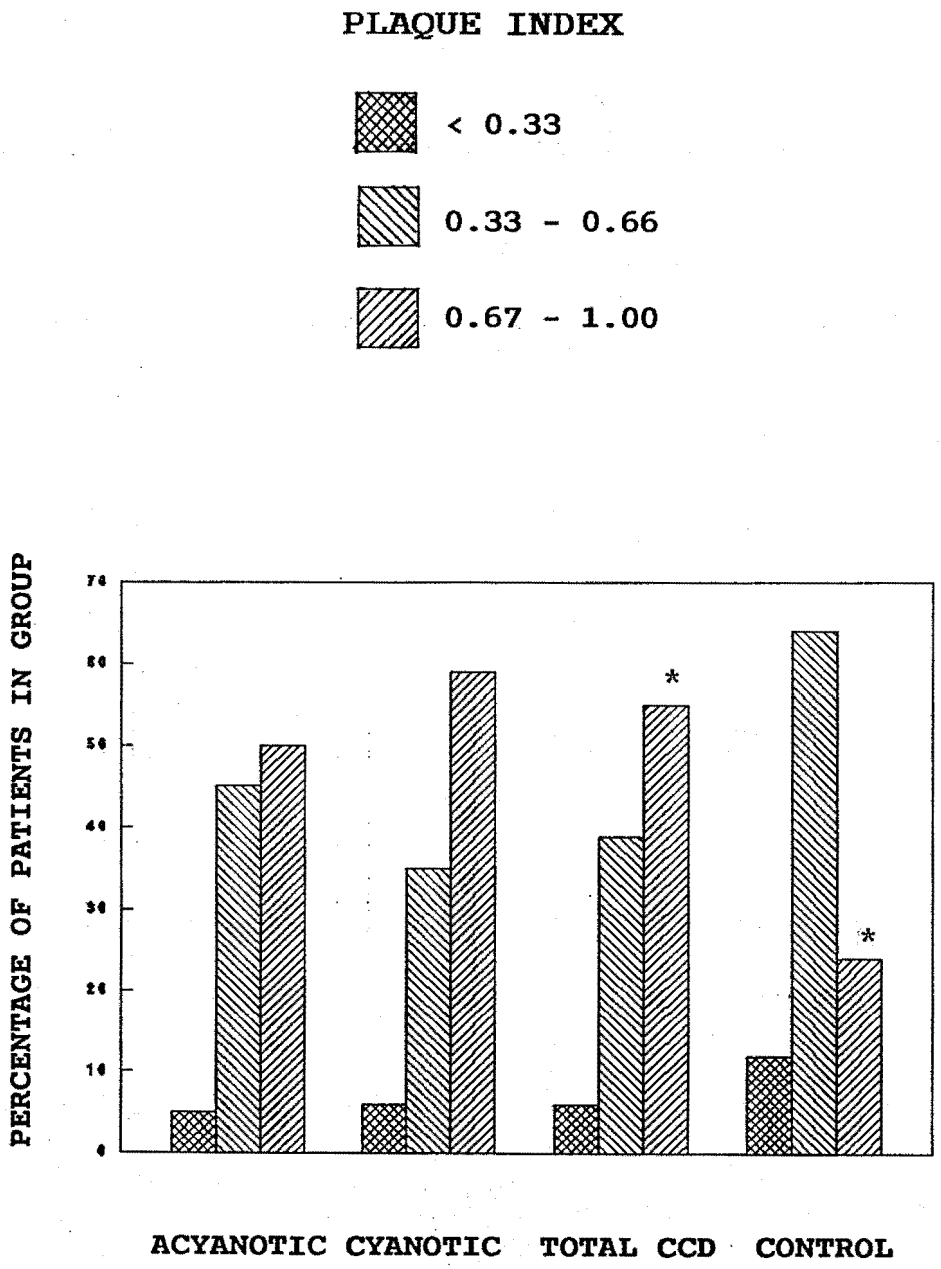

FIGURE 1. DISTRIBUTION OF PLAQUE INDICES OF CCD AND CONTROL PATIENTS

* Significant differences at $p < 0.01$.

DENTAL CARIES

Table 6A shows the dental caries index (dmft) for the primary teeth of the CCD and control patients. There was only a significant difference ($p < 0.01$) with regard to the decayed component (d) of the index between the Cyanotic (18%), total CCD (15%) and the control (10%) patients. This is illustrated graphically in Figure 2. Overall the mean dmft index for the CCD patients (4.2) was significantly greater ($p < 0.01$) than the dmft index for the control patients (2.3).

By contrast, no significant differences were found in the mean dental caries index for permanent teeth of CCD patients (0.9) and control patients (0.6) as shown in Table 6B. Also, the individual components of the dental caries index were not significantly different between the study and the control patients.

Table 6C shows that no significant differences were found in the percentages of caries-free children in the total number of dentitions in the CCD group compared to the control group. However, in children with primary teeth only there was a significantly greater percentage of caries-free children in the CCD group compared with the control group (26% vs 11%, $p < 0.01$). In the mixed dentition this was reversed with a significantly greater percentage of caries-free children in the control group as opposed to the CCD group (26% vs 6%, $p < 0.01$). The percentages of patients with caries-free dentitions at examination is illustrated in Figure 3.

TABLE 6A. DENTAL CARIES INDEX (dmft) FOR PRIMARY TEETH OF CCD AND CONTROL PATIENTS AT EXAMINATION

	Number of teeth affected				
Group	Acyanotic	Cyanotic	Total CCD	Total Control	p value
Teeth examined	n=303	n=210	n=513	n=414	
Teeth with open caries:(d)	39	40	79	41	< 0.01
Teeth extracted for caries:(m)	6	0	6	4	NS
Filled Teeth:(f)	31	15	46	47	NS
Total dmft	76	55	131	92	
No.patients	17	14	31	31	
Mean dmft	4.5	3.9	4.2	2.3	< 0.01

NS = NOT SIGNIFICANT
There were significant differences between CCD and control patients with regard to the proportion of decayed primary teeth present at examination and the overall Mean dmft.

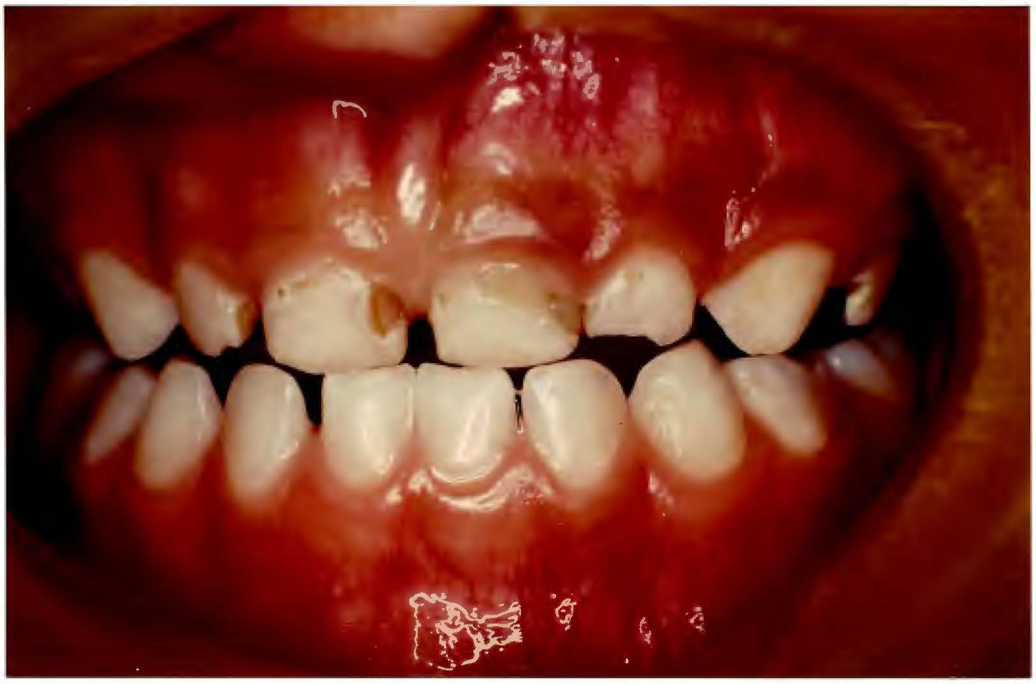

Illustration 6. Appearance of extensive dental caries in primary dentition of a patient with CCD.

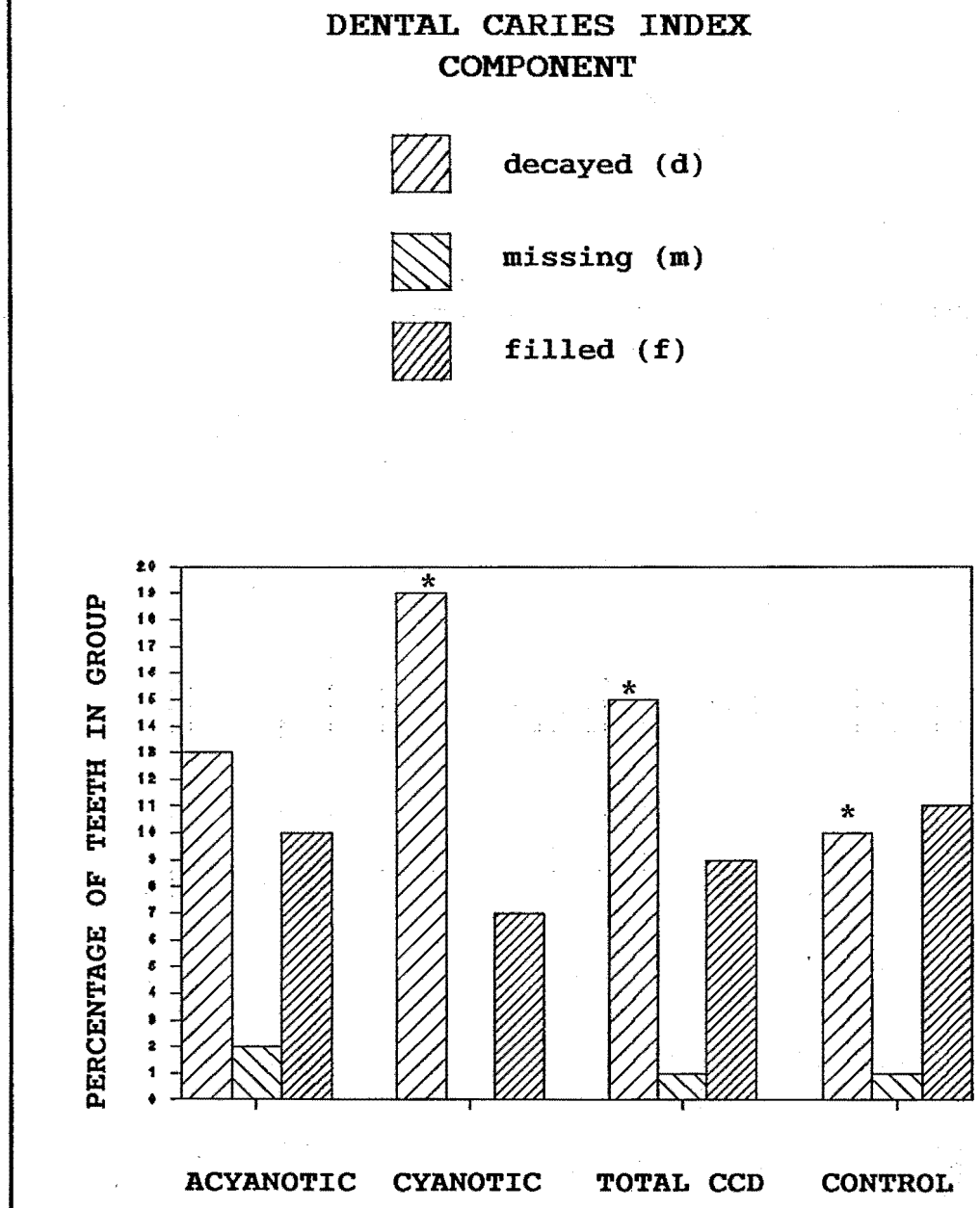

FIGURE 2. DISTRIBUTION OF DENTAL CARIES INDEX (dmft) OF CCD AND CONTROL PATIENTS

* Significant differences at $p < 0.01$.

TABLE 6B. DENTAL CARIES INDEX (DMFT) FOR PERMANENT TEETH OF CCD AND CONTROL PATIENTS AT EXAMINATION

	Number of teeth affected				
Group	Acyanotic	Cyanotic	Total CCD	Total Control	p value
Teeth examined	n=191	n=158	n=349	n=324	
Teeth with open caries:(D)	3	1	4	4	NS
Teeth extracted for caries:(M)	0	0	0	0	
Filled Teeth:(F)	9	6	15	15	NS
Total DMFT	12	7	19	19	
No.patients	11	10	21	25	
Mean DMFT	1.1	0.7	0.9	0.6	NS

NS = NOT SIGNIFICANT
There were no significant differences between CCD and control patients with regard to the proportion of permanent teeth affected at examination and the overall Mean DMFT.

TABLE 6C. NUMBER OF CCD AND CONTROL PATIENTS CARIES FREE AT EXAMINATION

	Number of Patients				
Group	Acyanotic	Cyanotic	Total CCD	Total Control	p value
Primary Dentition:					
n = 27	5	2	7	3	< 0.01
Mixed Dentition:					
n = 34	1	1	2	9	< 0.01
Permanent Dentition:					
n = 11	2	1	3	0	NS
Total					
n = 72	8	4	12	12	NS

NS = NOT SIGNIFICANT
There were significant differences between the CCD and control patients with regard to the number of caries free primary and mixed dentitions at examination.

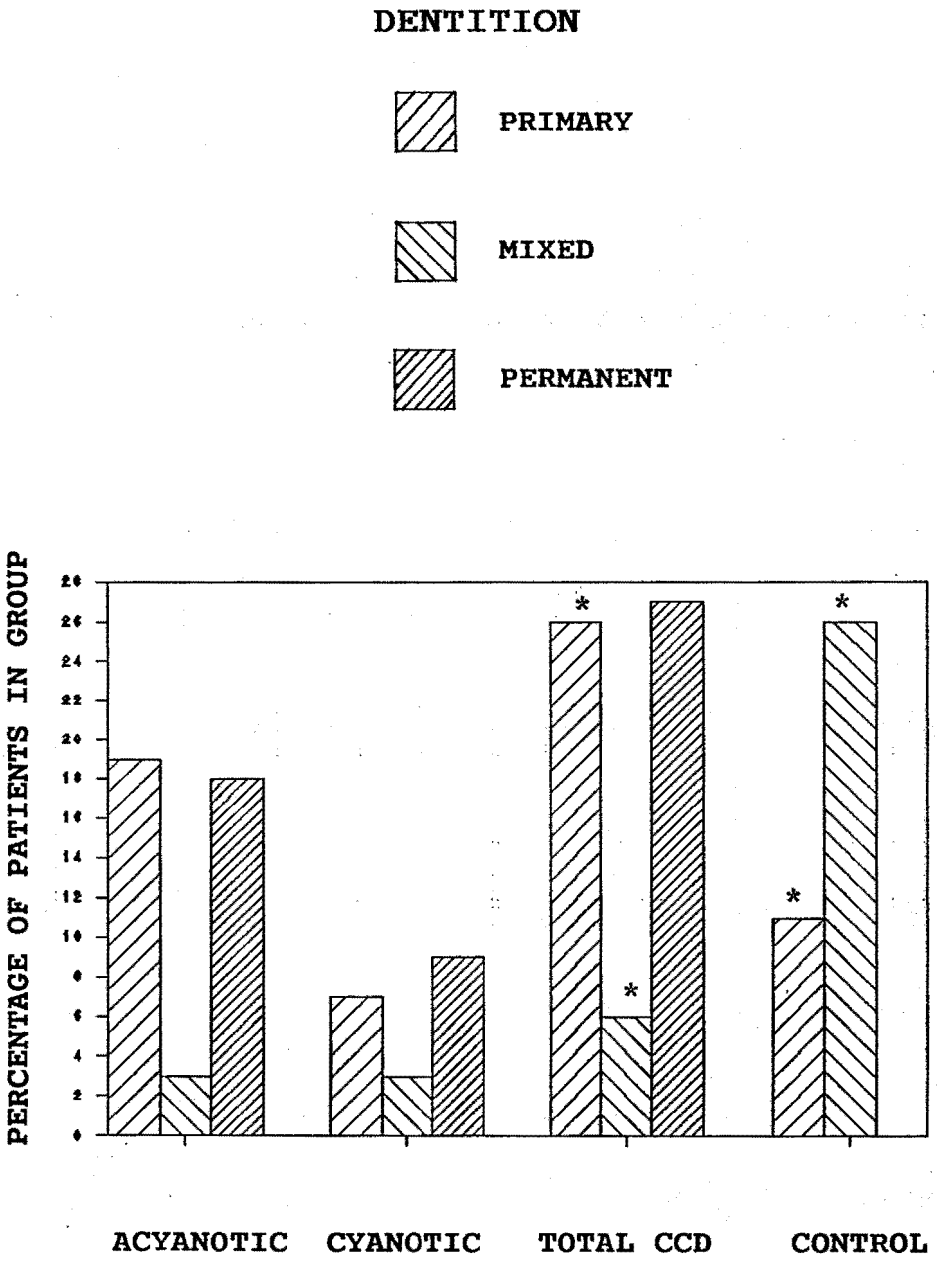

FIGURE 3. DISTRIBUTION OF CARIES FREE DENTITIONS OF CCD AND CONTROL PATIENTS

* Significant differences at $p < 0.01$.

DEVELOPMENTAL ENAMEL DEFECTS

The prevalence of developmental enamel defects of primary teeth is shown in Table 7A. Overall, there was a total of 16 (52%) CCD patients with developmental enamel defects of the primary teeth compared with only 7 (23%) in the control group ($p < 0.01$). The number of teeth affected was also significantly greater in the CCD group compared with the control group in regard to hypoplasia (4% vs 2%, $p < 0.01$) and opacity (13% vs 3%, $p < 0.001$) as shown in Table 7B. The distribution of enamel defects of primary teeth is shown in Table 7C which would indicate that the upper left incisors and lower second molars are the most commonly affected teeth. By contrast, the permanent teeth of the CCD group do not seem to be significantly different when compared to the control group. Proportionately, the prevalence of enamel defects and the number of teeth affected was observed to be similar in the CCD group (38% and 7% respectively) when compared to the control group (28% and 5% respectively). The data are shown in Tables 7D and 7E while Table 7F indicates the most commonly affected permanent teeth in the CCD patients are the upper left central incisor and the first permanent molars.

ENDODONTIC THERAPY PROVIDED

Table 8 shows that overall there were significantly greater ($p < 0.01$) numbers of patients with endodontically involved teeth in the CCD group compared to the control group. Although not significant, the numbers of teeth with untreated pulp pathosis and pulp capping were greater in the CCD group compared to the control group. Figure 4 shows that there were higher proportions of endodontically involved teeth in the Cyanotic (30%) and total CCD groups (21%) compared to the control group (9%), $p < 0.01$.

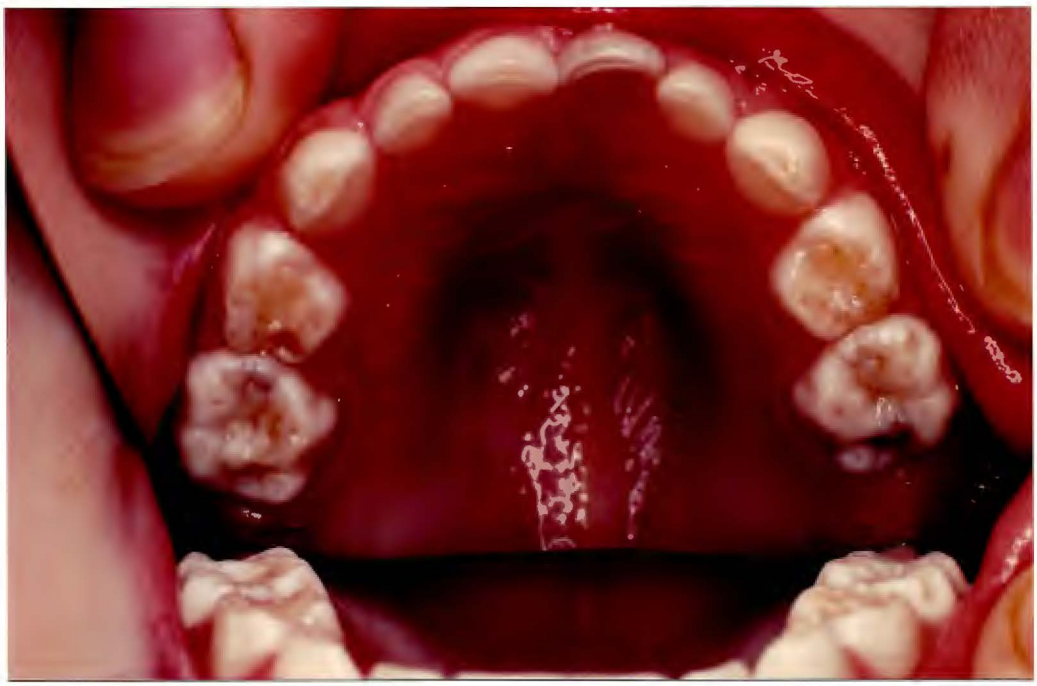

Illustration 7. Appearance of mild enamel hypoplasia and opacity in the maxillary primary dentition of a CCD patient.

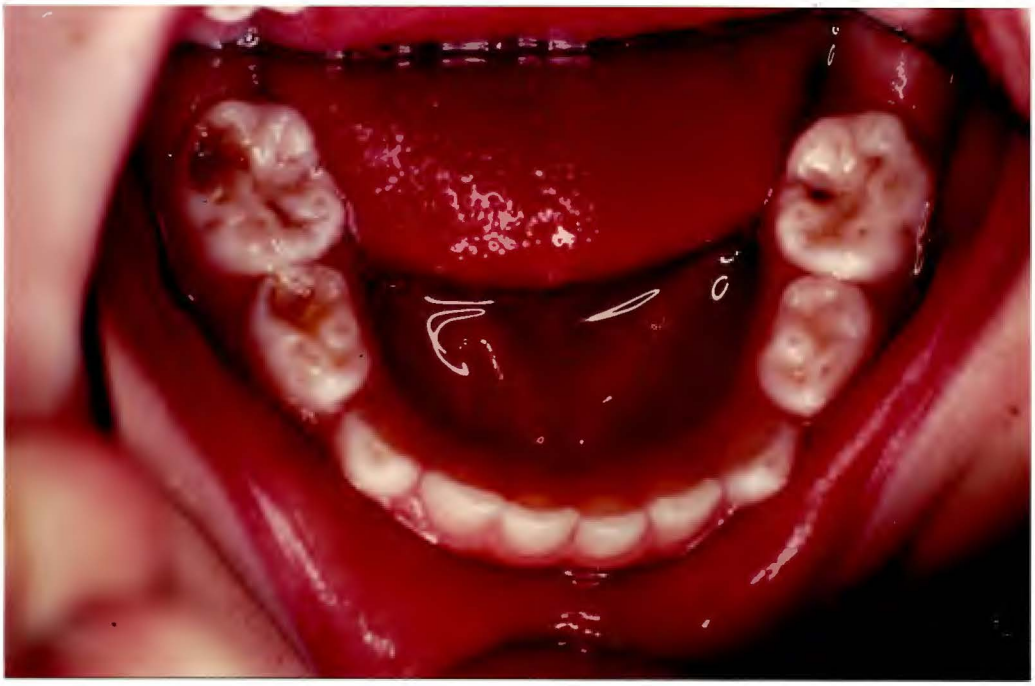

Illustration 8. Appearance of mild enamel hypoplasia and opacity in the mandibular primary dentition of a CCD patient.

TABLE 7A. PREVALENCE OF DEVELOPMENTAL ENAMEL DEFECTS OF PRIMARY TEETH IN CCD AND CONTROL PATIENTS

	Number of Patients with Primary teeth / (Percentage of total)				
Group	Acyanotic n=17	Cyanotic n=14	Total CCD n=31	Total Control n=31	p value
Hypoplasia:					
	2 (12)	4 (29)	6 (19)	1 (3)	< 0.01
Opacity:					
	5 (29)	5 (36)	10 (32)	6 (19)	NS
Total	7 (41)	9 (64)	16 (52)	7 (23)	< 0.01

NS = NOT SIGNIFICANT
There were significant differences between the CCD and control patients with regard to number of hypoplastic teeth in primary dentition and total number of enamel defects in the primary dentition.

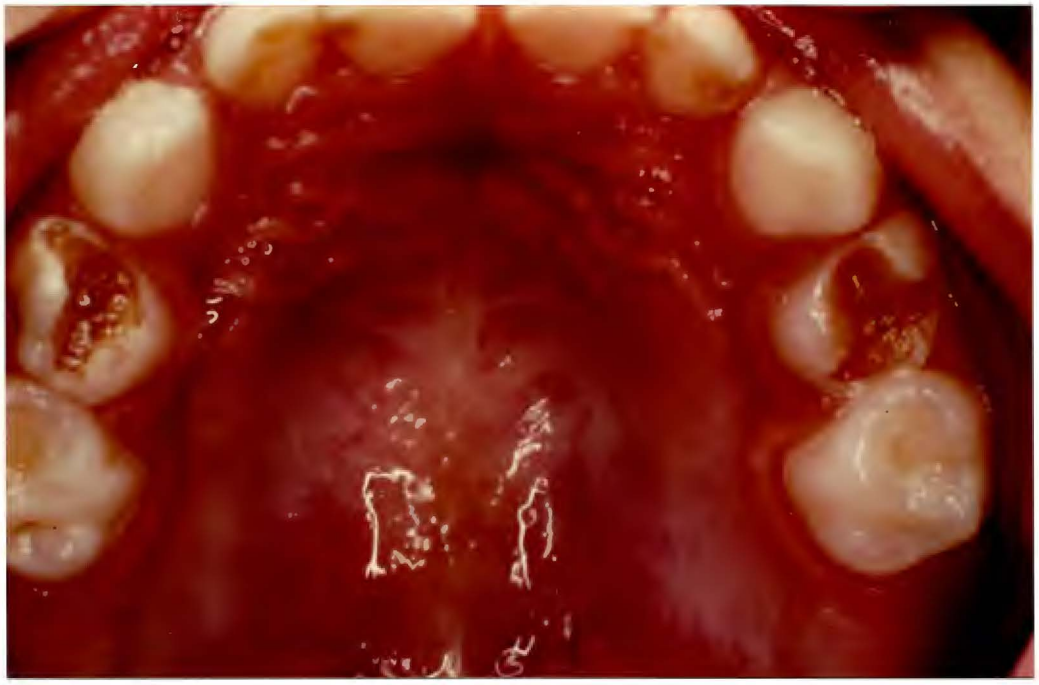

Illustration 9. Appearance of severe dental caries and opacity in the maxillary primary dentition of a CCD patient.

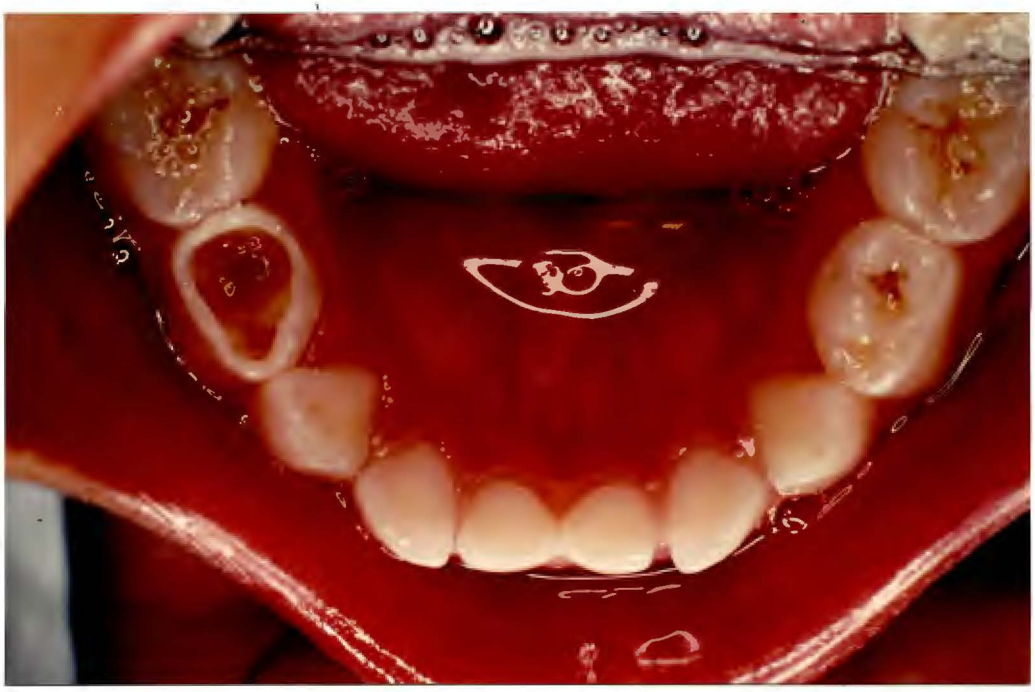

Illustration 10. Appearance of severe dental caries and opacity in the mandibular primary dentition of a CCD patient.

TABLE 7B. DEVELOPMENTAL DENTAL INDEX FOR PRIMARY TEETH OF CCD AND CONTROL PATIENTS AT EXAMINATION

	Number of primary teeth affected / (Percentage)				
Group	Acyanotic	Cyanotic	Total CCD	Total Control	p value
Teeth examined	n=303	n=210	n=513	n=414	
Hypoplasia:					
	5 (2)	17 (8)	22 (4)	9 (2)	< 0.01
Opacity:					
	19 (6)	50 (24)	69 (13)	11 (3)	< 0.001
Total	24 (8)	67 (32)	91 (17)	20 (5)	< 0.001

There were significant differences between the CCD and control patients with regard to the type of developmental enamel defect and the total number of defects in the primary dentition.

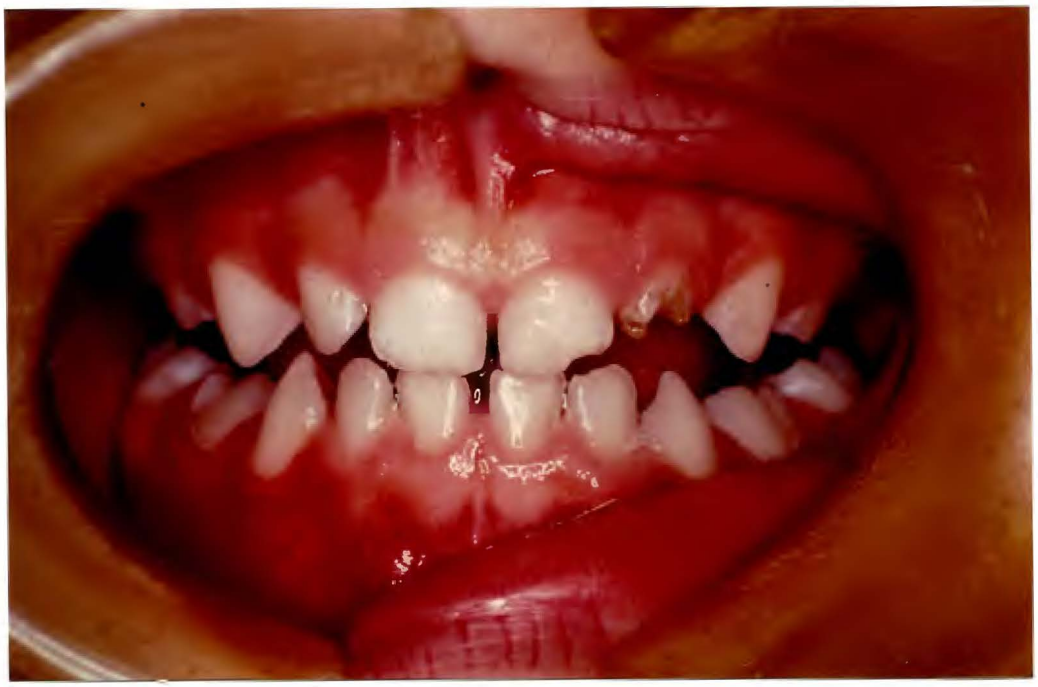

Illustration 11. Enamel defects were most commonly observed in the primary upper anterior teeth in patients with CCD.

TABLE 7C. DISTRIBUTION OF ENAMEL DEFECTS OF PRIMARY TEETH IN CCD PATIENTS

	Right					Left				
Maxillary	E	D	C	B	A	A	B	C	D	E
Hypoplasia:	3	2	0	0	1	4	1	0	2	2
Opacity:	2	3	3	4	3	3	3	3	3	2
Total	5	5	3	4	4	7	4	3	5	4
Mandibular	E	D	C	B	A	A	B	C	D	E
Hypoplasia:	2	2	0	0	0	0	0	0	1	2
Opacity:	6	6	2	3	3	4	3	2	6	5
Total	8	8	2	3	3	4	3	2	7	7

TABLE 7D. PREVALENCE OF DEVELOPMENTAL ENAMEL DEFECTS OF PERMANENT TEETH IN CCD AND CONTROL PATIENTS

Group	Number of Patients / (Percentage of total) with Permanent teeth				
	Acyanotic n=11	Cyanotic n=10	Total CCD n=21	Total Control n=25	p value
Hypoplasia:					
	0 (0)	1 (10)	1 (5)	1 (4)	NS
Opacity:					
	4 (36)	3 (30)	7 (33)	6 (24)	NS
Total	4 (36)	4 (40)	8 (38)	7 (28)	NS

NS = NOT SIGNIFICANT
There were no significant differences between CCD and control patients with regard to enamel defects in the permanent dentition.

TABLE 7E. DEVELOPMENTAL DENTAL INDEX FOR PERMANENT TEETH OF CCD AND CONTROL PATIENTS AT EXAMINATION

	Number of permanent teeth affected / (Percentage)				
Group	Acyanotic	Cyanotic	Total CCD	Total Control	p value
Teeth examined	n=191	n=158	n=349	n=324	
Hypoplasia:					
	0 (0)	1 (1)	1 (0)	1 (0)	NS
Opacity:					
	12 (6)	13 (8)	25 (7)	16 (5)	NS
Total	12 (6)	14 (9)	26 (7)	17 (5)	NS

NS = NOT SIGNIFICANT
There were no significant differences between CCD and control patients with regard to type of developmental enamel defect and total number of defects in the permanent dentition.

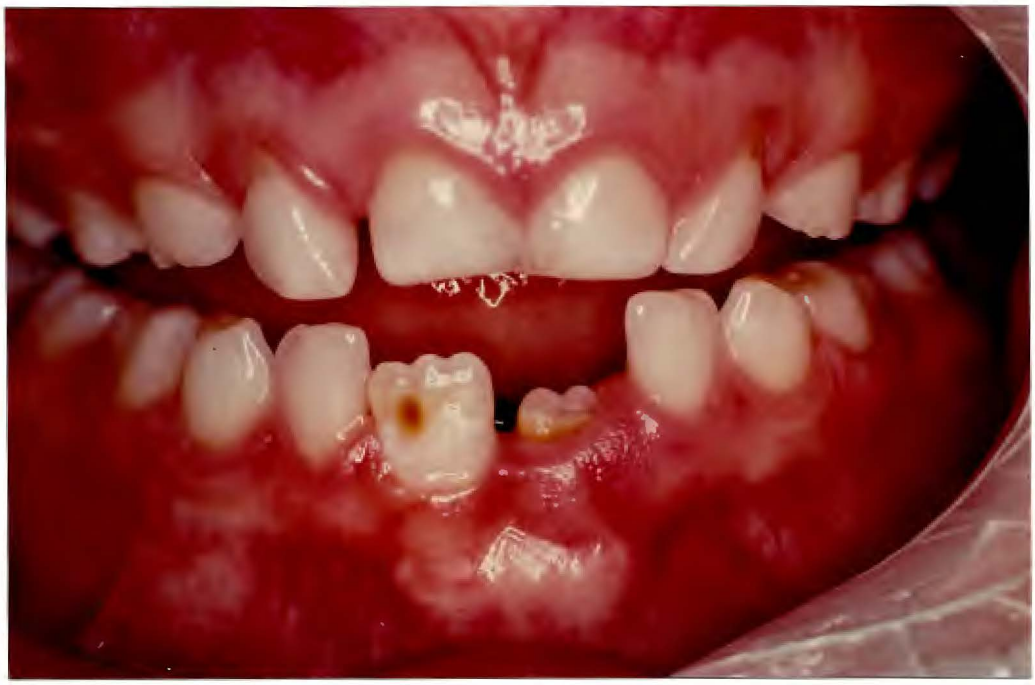

Illustration 12. Typical appearance of a permanent lower anterior tooth affected by enamel defects of a patient with CCD.

TABLE 7F. DISTRIBUTION OF ENAMEL DEFECTS OF PERMANENT TEETH IN CCD PATIENTS

	Right							Left						
Maxillary	7	6	5	4	3	2	1	1	2	3	4	5	6	7
Hypoplasia:	0	0	0	0	0	0	1	0	0	0	0	0	0	0
Opacity:	0	2	1	0	0	1	1	3	1	0	0	1	4	0
Total	0	2	1	0	0	1	2	3	1	0	0	1	4	0
Mandibular	7	6	5	4	3	2	1	1	2	3	4	5	6	7
Hypoplasia:	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Opacity:	1	4	0	0	0	0	0	1	0	0	0	0	4	1
Total	1	4	0	0	0	0	0	1	0	0	0	0	4	1

TABLE 8. TYPES OF ENDODONTIC THERAPY PROVIDED FOR CCD AND CONTROL PATIENTS

Group	Number of Patients: Acyanotic n=22	Cyanotic n=17	Total CCD n=39	Total Control n=33	p value
Pulp Capping:					
	2	2	4	2	NS
Pulpotomy:					
	0	1	1	0	NS
Root canal filling:					
	0	0	0	1	NS
Untreated pulp pathosis:					
	1	2	3	0	NS
Total	3	5	8	3	< 0.01

NS = NOT SIGNIFICANT
There were no significant differences between the CCD and control patients with regard to particular types of endodontic therapy, however there was a significant difference between the total number of patients receiving endodontic therapy.

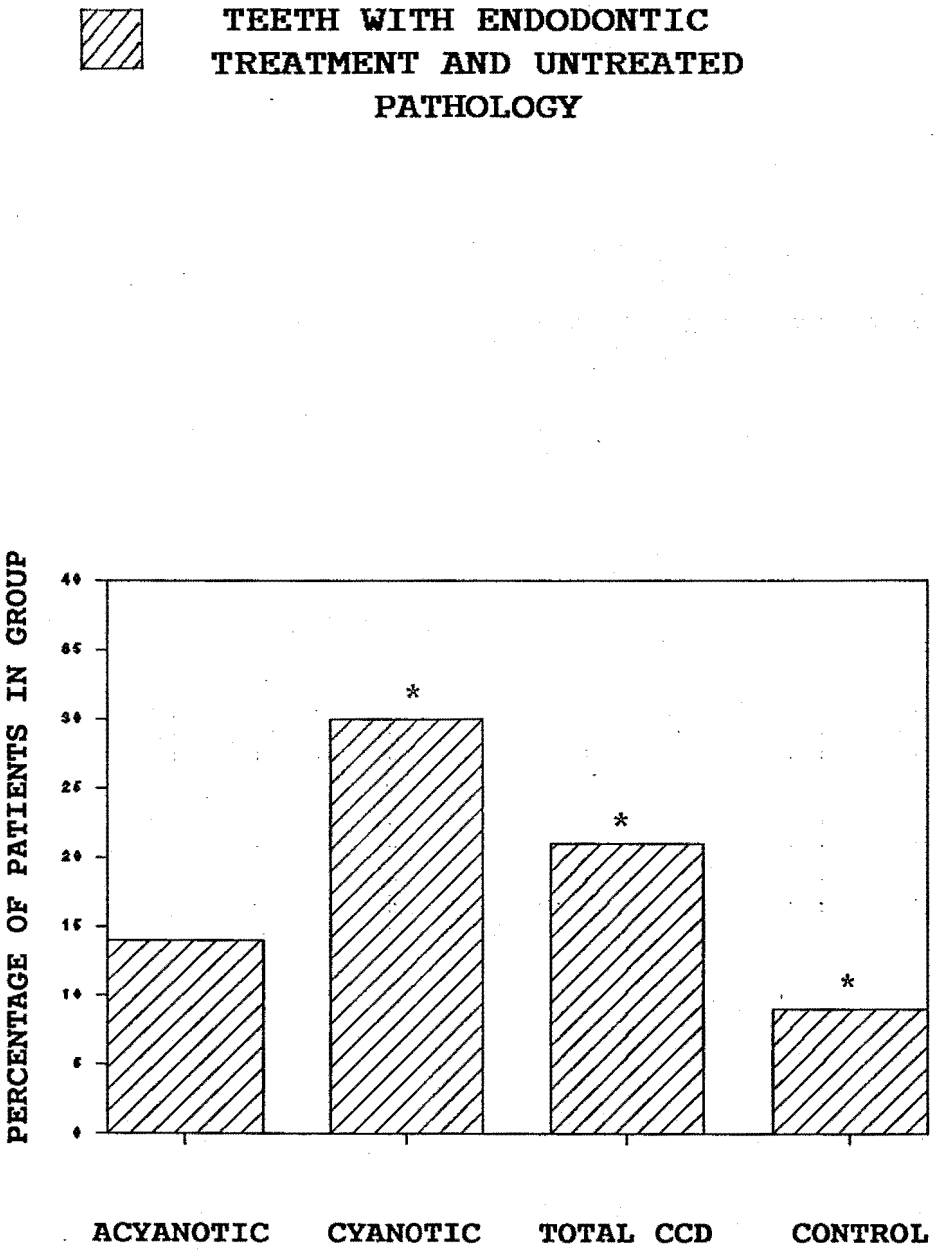

FIGURE 4. DISTRIBUTION OF ENDODONTICALLY INVOLVED TEETH OF CCD AND CONTROL PATIENTS

* Significant differences at $p < 0.01$.

TYPE OF OCCLUSION AND CROWDING

Thirty one (94%) of the control patients had a Class I occlusion, and 2 (6%) had a Class II occlusion (Table 9). By contrast, only 26 (67%) of the CCD group had a Class I occlusion while 11 (28%) had a Class II and 2 (5%) patients had a Class III occlusion. These differences in the prevalence of Class I and II occlusions are statistically significant ($p < 0.001$) and the percentages of patients in each group are illustrated graphically in Figure 5. Furthermore, there was also a higher prevalence of crowding in the total CCD group compared to the control group (31% vs 6%, $p < 0.001$). Anterior overjet was significantly greater than 2 mm in the total CCD group ($p < 0.001$) compared with the control group. This was not the case with anterior overbite, as there was no significant difference between the two groups.

DEVELOPMENTAL DENTAL ANOMALIES

Table 10 shows the prevalence of developmental dental defects in the study patients. The number of patients exhibiting these anomalies was low so that differences between the CCD and control groups were not significant.

TABLE 9. PREVALENCE OF OCCLUSION AND CROWDING IN CCD AND CONTROL PATIENTS

Group	Number of patients: Acyanotic n=22	Cyanotic n=17	Total CCD n=39	Total Control n=33	p value
Molar Relationship:					
Class I	15	11	26	31	< 0.001
Class II	6	5	11	2	< 0.001
Class III	1	1	2	0	NS
Crowding:	4	8	12	2	< 0.001
Anterior Overbite > 50%:	4	3	7	5	NS
Anterior Overjet > 2 mm:	6	5	11	2	< 0.001

NS = NOT SIGNIFICANT
There were significant differences between the CCD and control patients with regard to the molar relationship, anterior overjet greater than 2 mm and the presence of crowding.

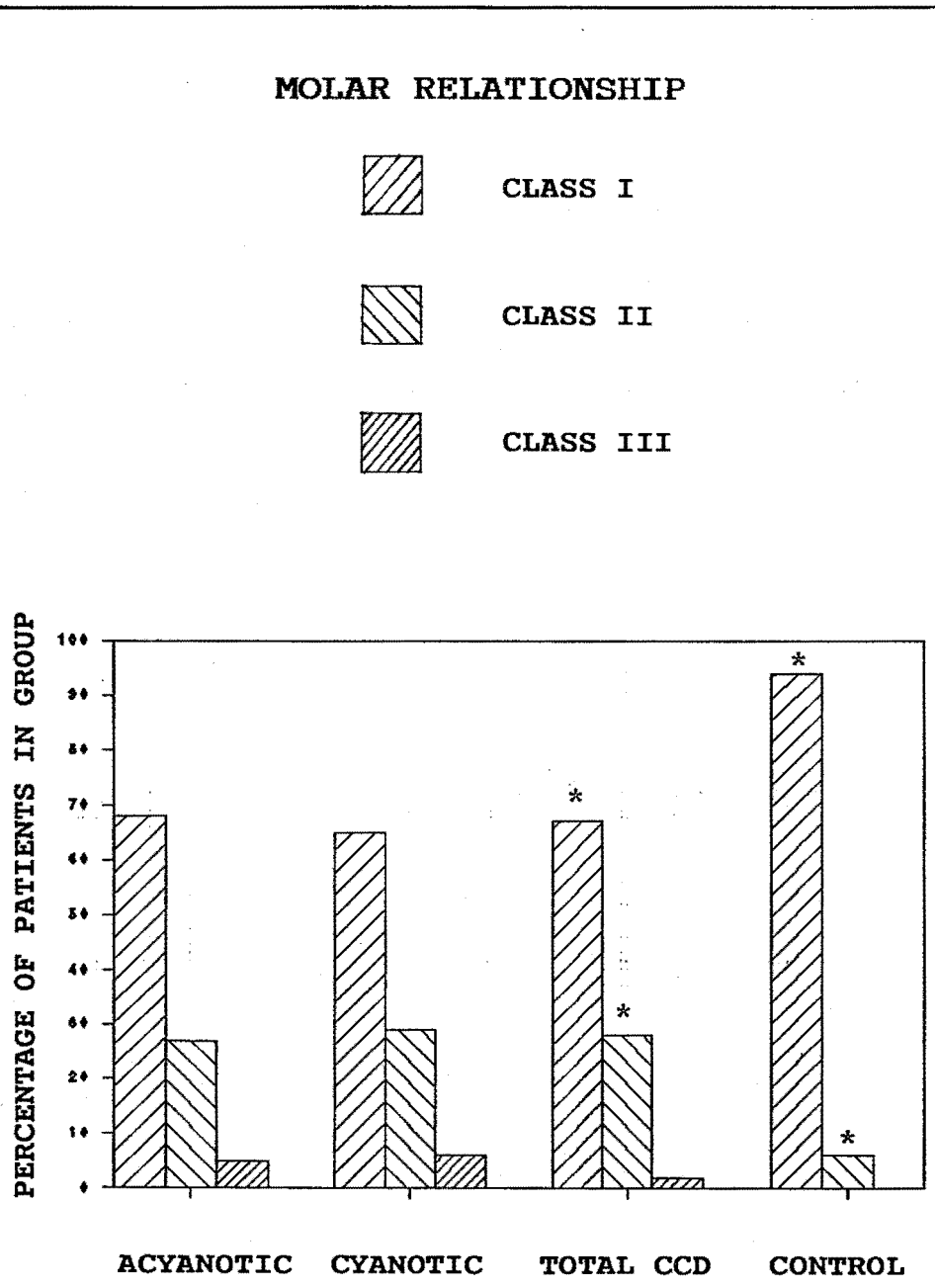

FIGURE 5. DISTRIBUTION OF OCCLUSION OF CCD AND CONTROL PATIENTS

* Significant differences at $p < 0.001$.

TABLE 10. PREVALENCE OF DEVELOPMENTAL DENTAL ANOMALIES IN CCD AND CONTROL PATIENTS

Group	Number of Patients / (Percentage of group) Acyanotic n=22	Cyanotic n=17	Total CCD n=39	Total Control n=33	p value
Hypodontia excluding 8's:	1 (5)	1 (6)	2 (5)	2 (6)	NS
Supernumerary:	0 (0)	0 (0)	0 (0)	0 (0)	
Microdontia:	0 (0)	1 (6)	1 (3)	0 (0)	NS
Macrodontia:	0 (0)	1 (6)	1 (3)	0 (0)	NS
Taurodontia:	0 (0)	1 (6)	1 (3)	0 (0)	NS

NS = NOT SIGNIFICANT
There were no significant differences between the CCD and control patients with regard to developmental anomalies.

PREVENTIVE DENTAL HEALTH BEHAVIOUR

As this study was conducted in Brisbane which does not have a fluoridated water supply, it was pertinent to examine fluoride supplement intake of the patients. This is shown in Table 11. Although the CCD group recorded a lower percentage (15% vs 21%) of children currently taking fluoride supplements, the differences were not significant. Parental attitudes to fluoride and previous fluoride exposure were similar in both groups.

With regard to oral hygiene habits, no significant differences between tooth brushing frequency and flossing were observed between the two groups. However, only 16 (41%) of CCD children received parental help with toothbrushing compared with 23 (70%) of control children ($p < 0.001$).

PROFESSIONAL PREVENTIVE CARE

Table 12 shows that 31% of children with CCD have had professional advice regarding the need for increased preventive dental health behaviour. Paradoxically, a smaller percentage received professionally applied topical fluoride therapy compared to control children (36% vs 48%), although the difference is not statistically significant. Advice on fluoride supplementation was received by equal percentages of CCD and control patients (Table 12). There were significantly more CCD patients who had received fissure sealants compared to control patients (21% vs 6%, $p < 0.001$). Similarly, the only significant difference in recall frequency was in the 6-12 month period with only 10% of CCD patients having visited a dentist within that period compared to 33% of control patients ($p < 0.01$).

TABLE 12. COMPARISON OF DENTAL PREVENTION PROVIDED BY PROFESSIONAL FOR CCD AND CONTROL PATIENTS

	Number of Patients / (Percentage of group)				
Group	Acyanotic n=22	Cyanotic n=17	Total CCD n=39	Total Control n=33	p value
Professional emphasis on need for increased prevention:	9 (41)	3 (18)	12 (31)	0	< 0.001
Topical Fluoride Therapy:	6 (27)	8 (47)	14 (36)	16 (48)	NS
Advice on Fluoride Supplements:	7 (32)	4 (24)	11 (28)	9 (27)	NS
Fissure Sealants:	7 (32)	1 (6)	8 (21)	2 (6)	< 0.001
Recall Frequency:					
< 6 months	11 (50)	9 (53)	20 (52)	13 (39)	NS
6-12 months	2 (9)	2 (12)	4 (10)	11 (33)	< 0.01
> 12 months	9 (41)	6 (35)	15 (38)	9 (28)	NS

NS = NOT SIGNIFICANT
The only significant differences between CCD and control patients were in regard to professional emphasis on the need for increased dental prevention, the placement of fissure sealants and recall frequency at 6-12 months.

ANTIBIOTIC PROPHYLAXIS REGIMENS

As shown in Table 13, 15 (38%) patients with CCD had not had previous dental treatment while 5 (21%) were not prescribed antibiotic prophylaxis prior to their dental treatment. Nineteen (79%) were prescribed a prophylactic antibiotic cover prior to dental treatment, amoxycillin (74%) being the most common, followed by penicillin V (21%). One patient was prescribed a combination of the above, while no patients were prescribed tetracycline or erythromycin. The most common route of administration was orally (89%) and the majority of patients (69%) were prescribed the antibiotics in dosages recommended by the Australian Dental Association Inc. (1989). Five patients (26%) were prescribed dosages of antibiotics below the ADA recommendation level while no patients were prescribed dosages above the ADA recommendation level. The preoperative time that the antibiotic was given prior to dental treatment varied from one hour for 7 (37%), to one day for 3 (16%), to greater than one day for 5 (26%) patients. Continuation of antibiotic prophylaxis after dental treatment also varied from no continuation for 4 (21%), one day after for 5 (26%), two to three days for 4 (21%), more than three days for 3 (16%) patients.

TABLE 13. TYPES OF ANTIBIOTIC PROPHYLAXIS REGIMENS PROVIDED TO CCD PATIENTS FOR DENTAL TREATMENT

Group	Number of patients / (Percentage of group) Acyanotic n=22	Cyanotic n=17	Total n=39
No previous dental treatment:			
n=15	9 (60)	6 (40)	15 (100)
No antibiotic prescribed for previous dental treatment:			
n=5	3 (60)	2 (40)	5 (100)
Antibiotics prescribed for previous dental treatment:			
n=19	10 (53)	9 (47)	19(100)
Amoxycillin	7 (37)	7 (37)	14 (74)
Erythromycin	0 (0)	0 (0)	0 (0)
Tetracycline	0 (0)	0 (0)	0 (0)
Penicillin V	2 (11)	2 (11)	4 (21)
Combination Penicillin V and Amoxycillin	1 (5)	0 (0)	1 (5)
Route of administration:			
Oral	8 (42)	9 (47)	17 (89)
Parenteral	2 (11)	0 (0)	2 (11)
Dose prescribed:			
ADA Recommendation*	7 (37)	6 (32)	13 (69)
< ADA Recommendation*	2 (11)	3 (16)	5 (26)
> ADA Recommendation*	0 (0)	0 (0)	0 (0)
Unknown	1 (5)	0 (0)	1 (5)

TABLE 13. CONTINUED

Preoperative time given before dental treatment:			
1 hour before	5 (26)	2 (11)	7 (37)
1 day before	0 (0)	3 (16)	3 (16)
> 1 day before	2 (11)	3 (16)	5 (26)
Unknown	3 (16)	1 (5)	4 (21)
Continuation after dental treatment:			
No continuation	2 (11)	2 (11)	4 (21)
6-8 hours after	0 (0)	0 (0)	0 (0)
1 day after	1 (5)	4 (21)	5 (26)
2-3 days after	3 (16)	1 (5)	4 (21)
> 3 days after	2 (11)	1 (5)	3 (16)
Unknown	2 (11)	1 (5)	3 (16)

* ADA = Australian Dental Association recommendation for antibiotic prophylaxis of Infective Endocarditis is :
Adults, 3.0 gm Amoxycillin 1 hour prior to dental procedure
Children 6-12 yrs, 1.5 gm Amoxycillin 1 hour prior to dental procedure
Children < 6 yrs, 750 mg Amoxycillin 1 hour prior to dental procedure

DIETARY SUGAR EXPOSURE

Comparison of mean daily sugar exposures in the diet of CCD and control patients revealed no significant differences (3.2 vs 2.9). Table 14 shows the data in relation to the sugar exposures obtained from analysis of the three day dietary charts.

MEDICATION INTAKE

Table 15 shows the medications and dietary supplements prescribed for the CCD and control patients. Twenty nine (76%) CCD patients received sweetened medications up to three times a day for control of their cardiac condition. In addition, sweetened antibiotics were prescribed to 25 (64%) CCD patients compared to 4 (12%) control patients for periods of greater than two weeks during childhood. Furthermore 7 (18%) CCD patients received daily multivitamin syrups in their early childhood. Consequently the number of daily sugar exposures from medications is significantly greater in the CCD group compared to the control group ($p < 0.01$).

When a comparison is made between the dental caries index and the medication intake of CCD patients, those patients receiving Digoxin or Digoxin and Diuretic twice daily had significantly higher numbers of decayed, missing and filled teeth compared to those not taking medications (mean 6.27 and 3.57 vs 2.67 respectively, $p < 0.01$). This is shown in Table 16 and the percentages of patients in each group are illustrated graphically in Figure 6. There were significantly higher proportions of patients with a dental caries index between 1 and 3 in the no medication and digoxin/diuretic groups compared to the digoxin only group ($p < 0.01$).

TABLE 14. COMPARISON OF THREE DAY SUGAR EXPOSURE BETWEEN CCD AND CONTROL PATIENTS

	Number of Exposures / (Percentage of total)				
Group	Acyanotic n=22	Cyanotic n=17	Total CCD n=39	Total Control n=33	p value
Sugar in Solution:					
Meal Times	30 (15)	34 (20)	64 (17)	51 (18)	NS
Between Meals	34 (17)	33 (19)	67 (18)	47 (17)	NS
Sugar in Retentive form:					
Meal Times	61 (30)	50 (29)	111 (29)	82 (29)	NS
Between Meals	80 (39)	57 (33)	137 (36)	104 (37)	NS
Total	205	174	379	284	
No.patients	22	17	39	33	
Mean/3 day sugar exposure	9.3	10.2	9.7	8.4	NS
Mean/ daily sugar exposure	3.1	3.4	3.2	2.9	NS

NS = NOT SIGNIFICANT
There were no significant differences between CCD and control patients with regard to mean three day dietary sugar exposure and mean daily sugar exposure.

TABLE 15. COMPARISON OF PREVIOUS MEDICATIONS AND SUPPLEMENTS PRESCRIBED FOR CCD AND CONTROL PATIENTS

Group	Number of patients / (Percentage) Acyanotic n=22	Cyanotic n=17	Total CCD n=39	Total Control n=33	p value
Cardiac medication (sucrose percentage^):					
Digoxin 2/Day (30)	7 (32)	10 (59)	17 (44)	0	< 0.01
Spironolactone 1/Day (20)	2 (12)	4 (24)	6 (16)	0	< 0.01
Chlorothiazide 1/Day (20)	3 (14)	3 (18)	6 (16)	0	< 0.01
Total	12 (58)	17 (100)	29 (76)	0	< 0.01
Other medication (sucrose percentage^):					
Antibiotics* (50-70)	17 (77)	8 (47)	25 (64)	4 (12)	< 0.01
Ventolin (55)	2 (12)	1 (6)	3 (8)	5 (15)	NS
Bricanyl (45)	1 (5)	1 (6)	2 (5)	0	NS
Aspirin (0)	1 (5)	4 (24)	5 (13)	0	< 0.01
Becotide (0)	2 (12)	0	2 (5)	0	NS
Others#	4 (18)	5 (29)	9 (23)	0	< 0.01
Dietary Supplement:					
Poly Joule+	0	3 (18)	3 (8)	0	NS
Calcium Gluconate	0	2 (12)	2 (5)	0	NS
Multi Vitamins	2 (12)	5 (29)	7 (18)	0	< 0.01

^ adapted from Bosso & Pearson, 1973 and Mater Childrens Hospital Pharmacy Department records.
* these include Amoxycillin, Bactrim, Cephlex and Erythromycin in the elixir form prescribed for longer than two weeks at a time.
# these include Beconase, Cardiprin, Inderal, Intal, Persantin, Verapamil and Warfarin which are only available in tablet form.
+ also known as Polycoze and contains 100% corn starch carbohydrate.

TABLE 16. ANALYSIS OF MEDICATION INTAKE AND DENTAL CARIES INDEX OF CCD PATIENTS

Group	Number of Patients			p value
	No Medication n=21	Digoxin Only n=11	Digoxin and Diuretic n=7	
Dental Caries Index:				
0	8	2	2	
1	4	0	1	
2	1	0	0	
3	2	2	2	
4	2	1	0	
5	0	1	0	
6	0	1	1	
7	1	0	0	
8	0	1	0	
9	2	0	0	
10	0	1	0	
11	1	1	0	
12	0	0	1	
19	0	1	0	
TOTAL	56	69	25	
No. of Patients	21	11	7	
MEAN ± SD	2.67 ± 3.5	6.27 ± 5.6	3.57 ± 4.3	< 0.01

NS = NOT SIGNIFICANT
There were significant differences between the group of patients taking Digoxin and the group not taking medication with regard to mean dental caries index.

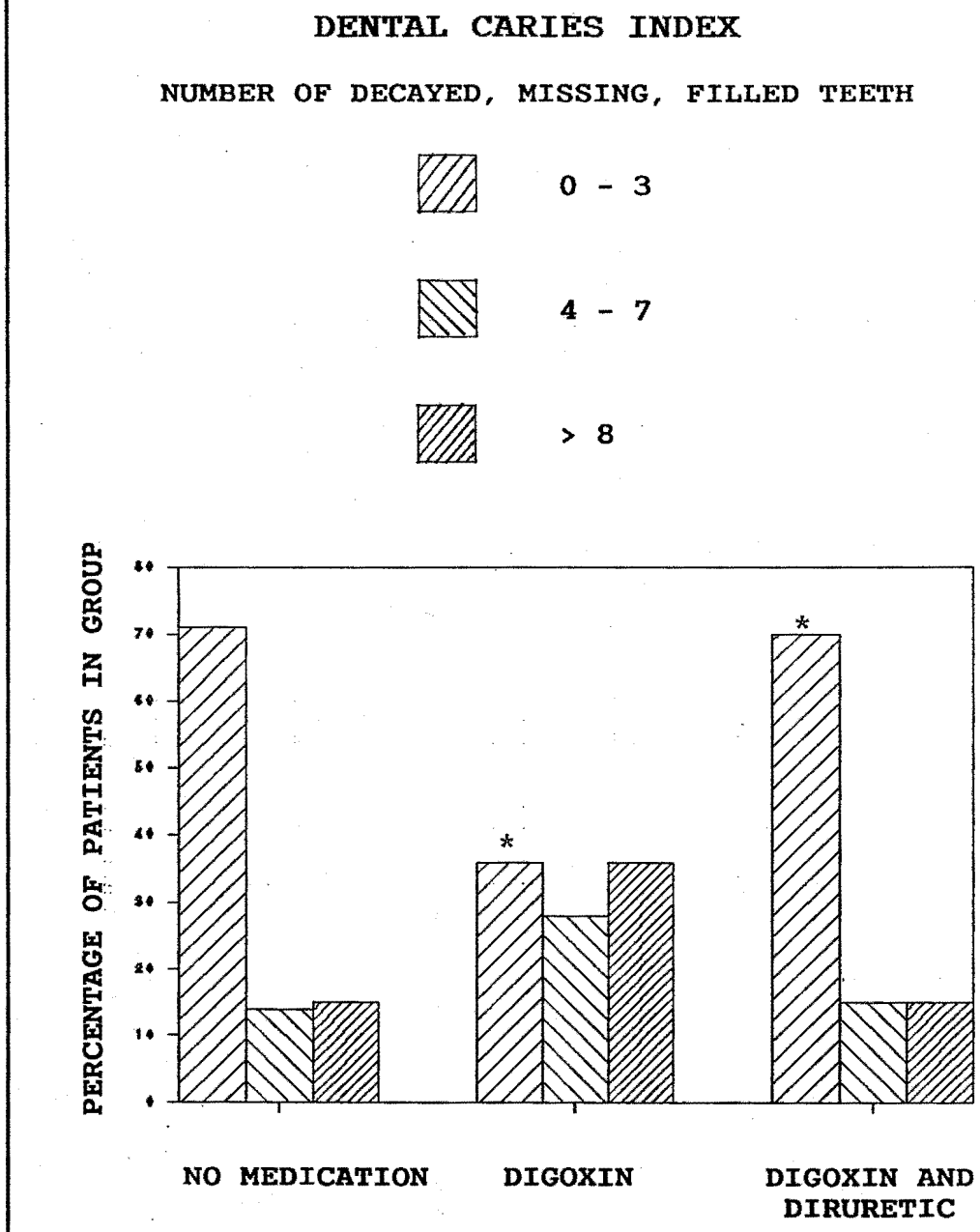

FIGURE 6. COMPARISON OF MEDICATION AND DISTRIBUTION OF DENTAL CARIES INDEX OF CCD PATIENTS

* Significant differences at $p < 0.01$.

DISCUSSION

In spite of the significance of congenital cardiac disease (CCD) in relation to the dental management of affected patients, there has been little previous research on the oral health and treatment requirements of these patients. The present study has shown that children with CCD suffer poorer oral health compared to control siblings. In the patients with primary dentitions, there were significantly greater numbers of teeth with untreated dental decay as well as endodontically involved teeth. In addition, there were twice as many children with developmental enamel defects in the group with CCD compared with controls. Furthermore, CCD children suffered significantly higher prevalence of Class II occlusion and crowding compared to control children.

The results of the present study extend those of the study of Berger (1978), over a decade ago, which also reported that children with cyanotic CCD had more actively carious teeth, lower levels of treatment as well as having less reminders to brush their teeth by mothers and had more cariogenic foods at home. The higher prevalence of enamel hypoplasia in the primary dentition shown in the present study may indicate a possible aetiological factor in the development of dental caries in the primary dentition.

A recent case report (Rockman, 1989) of a young child with Tetralogy of Fallot and medical reports (Thom & Howe, 1972; Holbrook et al., 1981; Rogers, 1989) of the dental status of various cardiac patients revealed numerous carious teeth, poor oral hygiene and cyanotic

gingivae similar to the clinical presentation of cyanotic CCD patients in this study. This poor oral health status may be due to many factors. Perhaps the parents become so involved with the medical problems that the oral health is given little or no attention at all. The present study shows that these children receive less parental help with toothbrushing and flossing compared with their healthy siblings.

The importance of home care must be constantly stressed. Since the magnitude of these children's medical problems complicates the most routine of dental procedures, prevention of disease must be the common goal of the dentist and parent. Yet, the present study showed that only 31% of these children received professional advice on the need for increased preventive dental care. Also the prevalence of professional preventive therapies such as advice on fluoride supplementation (28%), topical fluoride application (36%) and placement of fissure sealants (21%) was low.

The low levels of fluoride supplementation in both groups (15-21%) of children reflected previous observations (Seow et al., 1987) in Brisbane which has no water fluoridation. When parents were questioned regarding their attitudes towards fluoride, up to 87% gave a positive response yet this favourable attitude did not bring about an active behavioral change in providing fluoride supplements to their children. It would seem that up to 41% of parents gave their children with CCD fluoride supplements during infancy but that this percentage dropped to 15% when the child grew older. The reason for this change in attitude and behaviour was not determined and would require further investigation.

Increased prevalence of developmental defects such as facial anomalies, malocclusion and cleft palate are likely to be related to environmental and hereditary factors associated with CCD. The association of congenital conotruncal cardiac abnormalities and facial dysmorphism (Radford, 1985; Bell et al., 1990) suggests that these defects may have common embryopathic origins related to abnormal neural crest tissue behaviour involving first and fourth branchial arch maldevelopment. Embryological insult between the fourth and seventh weeks of gestation is also indicated. This may influence enamel development of the primary dentition and result in the increased prevalence of enamel hypoplasia as revealed in the present study. It is also possible that enamel hypoplasia in CCD children may have resulted from systemic disturbances associated with the cardiac disease during antenatal and neonatal development (Seow et al.,1987; Seow et al., 1989). The present study has shown that these children suffered more antenatal complications and underwent more neonatal surgery than healthy siblings ($p < 0.01$). Severe metabolic derangements associated with neonatal surgery may adversely affect enamel formation in the primary dentition.

Increased prevalence of dental caries in children with CCD is likely the result of increased tooth susceptibility from developmental enamel defects as well as from chronic intake of sweetened medications (Lokken et al., 1975; Roberts & Roberts, 1981; Feigal et al., 1981 & 1984). Seventy six percent of CCD patients in the present study were taking Digoxin and Diuretic syrup preparations containing 20-30 percent sucrose (Bosso & Pearson, 1973). Furthermore, seven (18%) were taking vitamin syrup supplementation containing sucrose and three (8%)

had taken high caloric supplementation containing dextrose in the form of corn starch thus increasing the frequency of daily sugar exposure to the teeth. Although it is also possible that CCD children were further indulged in more frequent sweet snack foods compared to control children, analysis of food items from diet histories did not reveal such likely increases in sugar exposures.

The low levels of oral health in children with CCD compared to control children may have significant implications in the medical management of these patients. Large untreated carious lesions may quickly progress to dental pulp pathoses which are associated with bacteraemia and potential infective endocarditis (IE) and brain abscess formation.

Certain immunodeficiency syndromes have a high association with CCD. Humoral and cell mediated deficiency have both been reported (Radford et al., 1986; Radford et al., 1990) resulting in an increased susceptibility to infection. The infection-proneness of these children may be explained in part by mechanical/haemodynamic factors such as bacterial endocarditis developing on valvular lesions, pulmonary infections from compression of bronchi by engorged vessels and brain abscess from right-to-left shunts. Underlying immunodeficiency disorder affecting T cells and IgG subclasses may also explain frequent occurrence of systemic infection as shown by the increased frequency (64% vs 12%) of long term antibiotic medication prescription to the CCD group in the present study.

Poor dental management may further compromise oral health in CCD patients. Prevention of IE is essential in patients with predisposing CCD. The proper regimen of antibiotics is well publicised by the Australian Dental Association Inc. (1989). It is important to investigate the child's previous antibiotic experience, since frequent exposures to antibiotics may have resulted in the development of resistant microbial strains (Leviner et al., 1987). Medical consultation should be sought to assure the proper choice and dosage of prophylactic antibiotics. It is also interesting to note that no prior oral chlorhexidine antisepsis was employed by dentists in the prevention of IE even though a recent study (Tzukert et al., 1986) has shown the beneficial effect of such procedures in reducing the severity of antimicrobial counts prior to oral surgical manipulations.

The sequelae of infection in patients with CCD are such that the dentist may have to reevaluate some of the standard and acceptable clinical procedures in dentistry. Because of the uncertainty of certain types of endodontic therapy such as pulp capping and pulpotomy which can lead to chronic pulpal inflammation, these procedures are usually not recommended in patients with CCD. It is my opinion that pulpectomies are also contraindicated in these patients since the root canal system is so diverse in primary teeth it may be difficult or impossible to debride an infected tooth completely. This can result in a focus of infection and place the patient at risk. Valachovic & Hargreaves (1979) reported on a child with CCD in whom a culture from a brain abscess contained flora similar to that found in the oral cavity. In that case it was speculated that a primary molar in which

a pulpectomy had been done three years previously was the focus of infection.

In the present study, a total of 8 (21%) of CCD patients had either untreated pulp pathoses or pulp therapy on primary teeth indicating that in these children there are high possibilities of oral foci of infection and potential IE. Perhaps extraction and space maintenance are the more appropriate treatments in children with CCD.

The present study has also shed light on the antibiotic prophylaxis regimens employed by dentists for the prevention of IE. It is disturbing to note that five (13%) of CCD patients did not receive prophylactic antibiotics prior to dental treatment. Yet the incidence of bacteraemia following dental procedures has been well documented (Burket & Burn, 1937; Jones et al., 1970; Lineberger & De Marco, 1973; Farrington, 1973; Faigel & Gaskill, 1975; De Leo et al., 1977; Baltch et al., 1977).

Of the patients that received prophylaxis, most (74%) were prescribed amoxycillin, the antibiotic currently recommended by the Australian Dental Association Inc.(1989) and in the correct dosages. Penicillin V was prescribed in four cases (21%) even though the superiority of amoxycillin has been demonstrated conclusively (Shanson et al., 1978 & 1980). The standard regimen for oral antibiotic use specifies amoxycillin as the drug of choice. Amoxycillin is absorbed more rapidly and completely from the gastrointestinal tract than penicillin V. The ADA also recommends dose ranges for children at various ages. The pediatric dose obviously should not exceed the adult dose of three

grams one hour prior to the dental procedure. They also recommend that penicillin V is a rational and acceptable choice for prophylaxis following dental procedures. However, amoxycillin has been shown to be unsuccessful in preventing IE in a number of cases (Durack et al., 1983; Denning et al., 1984)

It is notable that eight (42%) of CCD patients had prophylactic antibiotics prescribed as early as one day or more, prior to dental treatment. This practice is contradictory to the current recommendations of all health authorities (European Society of Cardiology, 1985; Medical Letter, 1987; British Society for Antimicrobial Chemotherapy, 1990; Council on Dental Therapeutics : American Heart Association, 1991) which state that the prophylactic antibiotic regimen need only be given one hour prior to dental treatment. Preoperative prophylaxis is likely to be more effective when given in sufficiently high doses just prior to the procedure in order to ensure effective serum concentrations of the antibiotic. Also continuation of antibiotic prophylaxis after dental treatment was variable with seven (37%) patients receiving antibiotics for more than one day post treatment. To guard against the development of resistant strains it is recommended that the antibiotic should be used for a relatively brief postprocedural period. These variations from the current recommendations for antibiotic prophylaxis were also observed in other studies (Brooks, 1980; Sadowsky & Kunzel, 1984; Murrah et al., 1987; Kryshtalskyj, 1988; Gould, 1990; Gutschik & Lippert, 1990).

Infective endocarditis can occur in spite of prophylactic measures so dentists and physicians should maintain a high level of suspicion

about any unusual clinical course. As two children within the study group gave a history of IE previously this complication should always be considered after dental treatment. The health authorities emphasise that these guidelines exist to assist practitioners in the exercise of their own clinical judgement regarding individual cases. The recommendations are not intended as a universal standard of care. Antibiotic prophylaxis is indicated for all dental procedures likely to cause gingival haemorrhage. This does not include normal exfoliation of deciduous teeth, injection of local anaesthetic except for periodontal ligament injections or simple adjustment of orthodontic appliances. Also included in this list are restorations above the gingival margin, however placement of a rubber dam for these procedures would likely necessitate prophylaxis. Endotracheal intubation does not require prophylaxis in the view of some authors (Council on Dental Therapeutics: AHA, 1991) unless it is accompanied by some other invasive procedure.

The risk of bacterial endocarditis after an exposure of low-grade bacteraemia caused by vigorous tooth-brushing or chewing is undoubtedly less than the intense bacteraemia caused by oral surgical procedures (Lineberger & De Marco, 1973; Faigel & Gaskill, 1975; Guntheroth, 1984). However, the cumulative risk of daily low-grade bacteraemia places patients with severe CCD at risk. This study has shown that the CCD group had significantly more patients with higher plaque scores (0.33-1.00) than their healthy siblings ($p < 0.01$). Hence they may be at higher risk for development of bacteraemia than the control patients from everyday oral hygiene procedures and

mastication. Maintaining better oral hygiene will decrease the risk of daily bacteraemias and prove extremely beneficial in preventing IE.

Elliott (1975) described a special coordinated dental service for children with CCD within the Department of Child Dentistry, Queen's University, Belfast. The aims of this service were to ensure that each child received all necessary dental advice from the time of the condition being first diagnosed, provision of a team of pedodontists and anaesthetists skilled in dealing with these children and provision for ongoing study and research. A pedodontist attends all sessions of the pediatric cardiology clinics to provide oral health counselling and preventive dental care. Where corrective cardiac surgery is planned, children have a dental check two months before the operation. The aim of this check is to avoid the need for any but the most minor dental treatment for two months prior and two months following surgery. Where dental treatment is necessary, this is undertaken by the child's family dentist, as an hospital out-patient or in-patient depending on the severity of the cardiac and dental condition. The children were recalled at regular four monthly intervals for examination and treatment.

This study has shown the need for such a specialised service here in Queensland due to the poor standard of dental care provided to this group of children. As improvements in medical technology advances the life expectancy of these children well into adulthood, it is the responsibility of the dental professional to provide a continuing high standard of care to further enhance their quality of life.

In conclusion, this study has shown that children with congenital cardiac disease suffer poorer oral health compared to control children. This is most likely related to abnormal dental formation as well as to poor oral hygiene. Furthermore, a significant number of these children did not receive optimal professional dental care, and only a minority were told about the increased need for dental prevention. It is hoped that the present study has highlighted the oral problems encountered in children with congenital cardiac disease and emphasised the need for vigorous preventive care in these special patients.

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THE UNIVERSITY OF QUEENSLAND
DEPARTMENT OF DENTISTRY

Dental School
Turbot Street
BRISBANE QLD 4000
AUSTRALIA

APPENDIX 1

Dear

Thank you for accepting the invitation to participate in my dental research project. At the appointment, I will examine your child's teeth and advise you of any treatment necessary. If you so wish further treatment can be undertaken at the Dental School.

Could you please bring a brother or sister (if there is one) to participate in the study, as I would like to make a comparison between the children's dental health.

Thank you once again for bringing your child to the Dental School.

Yours sincerely,

Kerrod Hallett.

Telephone:
National: (07) 365 8111
International: +617 365 8111

Fax:
National: (07) 365 8199
International: +617 365 8199

NAME: SEX:

APPENDIX 2

ADDRESS: DATE OF BIRTH:

DATE OF EXAM:

AGE AT EXAM:

MATERNAL HISTORY

Maternal age at birth:

F in pregnancy - F Water:
Supplements:

Pregnancy complications (including infections):

Birth complications:

Medications during pregnancy:

Smoking during pregnancy:

Alcohol during pregnancy:

Dental check-up during pregnancy:

NEONATAL HISTORY (1st 30 days)

Birth weight:

Gestational Age:

Intubation:

Neonatal Complications:

Surgical Treatment:

Medications:

POST NATAL HISTORY

I. MEDICAL

1. Diagnosis of Cardiac Defect:

2. Medical Syndrome?

3. Other Medical Conditions:

4. Medications (Current):

5. Immunizations; Measles, Chickenpox, Rubella, Mumps.

II. DENTAL

1. Family Dentist:

2. How long since last check up:

3. Regular visits in the past:

4. Types of previous treatment:

   Restorative Extractions Preventive Perio Ortho Other

5. Form of management (L.A./R.A./G.A.)

6. Any complications with above dental treatment:

7. (a) Ever been covered by antibiotics for dental work:

   (b) If yes, for what procedures:

   (c) What antibiotics have been given for prophylaxis:

      i) Dose :

      ii) Route :

      iii) How many times/day:

8. Fluoride History -

   (a) Ever taken F before? Against F: When started -
   Who advised -

   (b) In what form:

   (c) How regular:

   (d) Topical F therapy:

   (e) Lived in F^{ed} area:

9. Preventive Dental Health Behaviour

Tooth brushing -

(a) How many times/day:

(b) Does parent help:

Flossing -

(a) Does child floss:

(b) How often:

(c) Parental help:

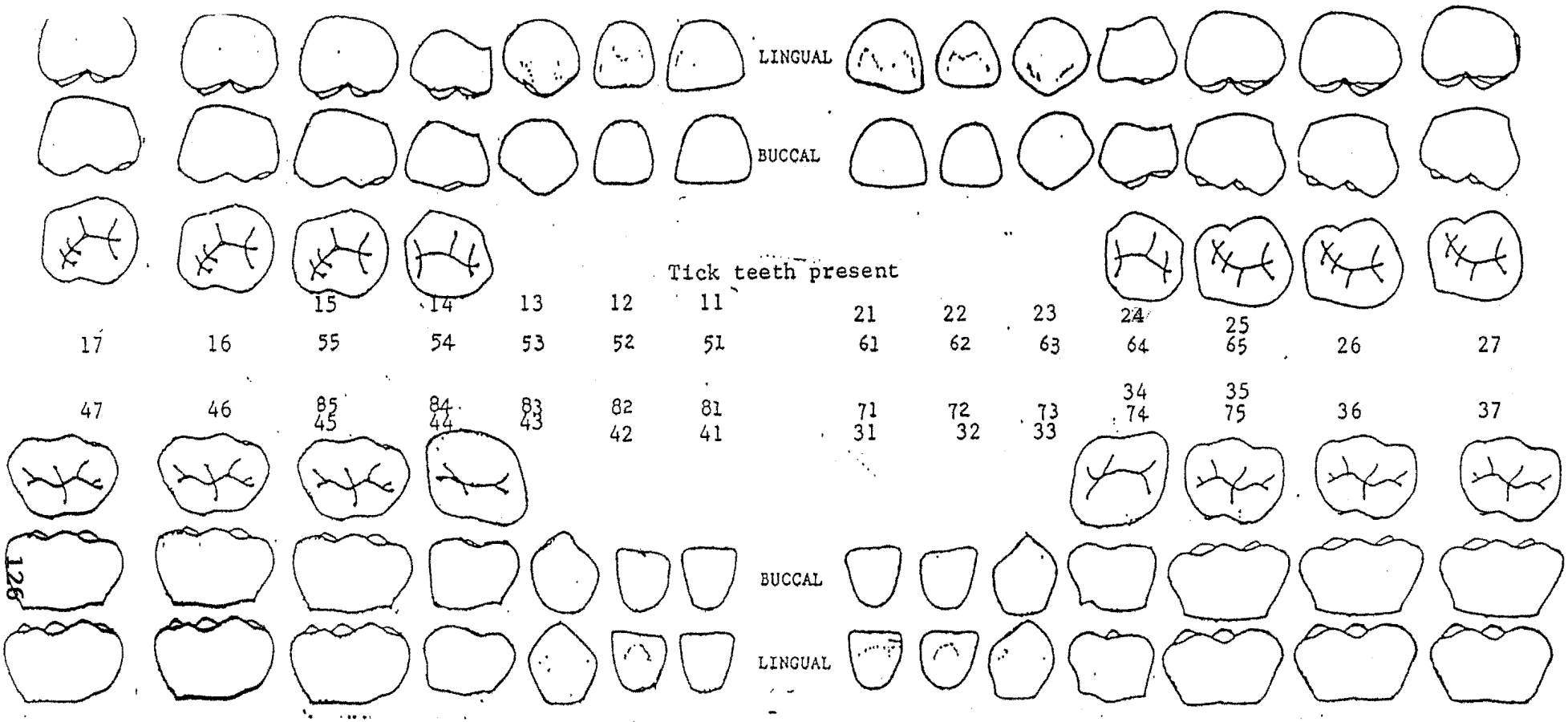
10. Has anyone explained need for increased dental prevention:

11. Sugar Exposures (analysis of diet charts)

Mean daily sugar exposures:

Day	Solution Form	Retentive Form	Total
1			
2			
3			
TOTAL:			

Mean:



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C = Caries (Red) 1. White Spot 2. Cavitation

O = Opacities (Blue) White/brown Intact

H = Hypoplasia (Pencil)

Crown Form

Agenesis of teeth

Occlusion

Molar relationship

Anterior

PLAQUE & GINGIVAL INDICES

Date ___/___/___

Plaque Index

16(55) 21(61) 24(64) 36(75) 41(81) 44(84)

Total scores / Total surfaces = P.I.

Some helpful suggestions in completing this FOOD DIARY:

1. Choose THREE successive days.
2. Make at least ONE of these days a Saturday or a Sunday.
3. Record EVERYTHING that you eat or drink.
4. Provide as much INFORMATION as possible.
5. Describe AMOUNTS in everyday terms such as cup, tablespoon, slice etc.

REMARKS and RECOMMENDATIONS:

FOOD GROUPS		MINIMUM DAILY SERVINGS (MDS)	EVALUATION: SERVINGS PER DAY DAY 1	DAY 2	DAY 3	SUMMARY Average	Average minus MDS
MILK and CHEESE	CHILDREN	3					
	TEENS	4					
	ADULT	2					
MEAT and OTHER BODY BUILDERS	EVERYONE	2					
FRUIT and VEGETABLES	EVERYONE	4					
BREAD and CEREALS	EVERYONE	4					
FAT							
FLUIDS							

SUGAR FREQUENCY		NUMBER PER DAY DAY 1	DAY 2	DAY 3	SUMMARY TOTAL	Average per Day
IN SOLUTION	During meals					
	End of meals					
	Between meals					
IN RETENTIVE FORM	During meals					
	End of meals					
	Between meals					

FOOD DIARY

and

DIET EVALUATION

PATIENT

DATE

Your Dentist: _____

THE UNIVERSITY OF QUEENSLAND
DENTAL SCHOOL
TURBOT ST.
BRISBANE. Q. 4000

APPENDIX 3

AY 1: _ _ / _ _ / _ _	AMOUNT	DAY 2: _ _ / _ _ / _ _	AMOUNT	DAY 3: _ _ / _ _ / _ _	AMOUNT
EFORE REAKFAST					
REAKFAST					
ETWEEN EALS					
UNCH					
ETWEEN EALS					
EVENING MEAL					
UPPER					